

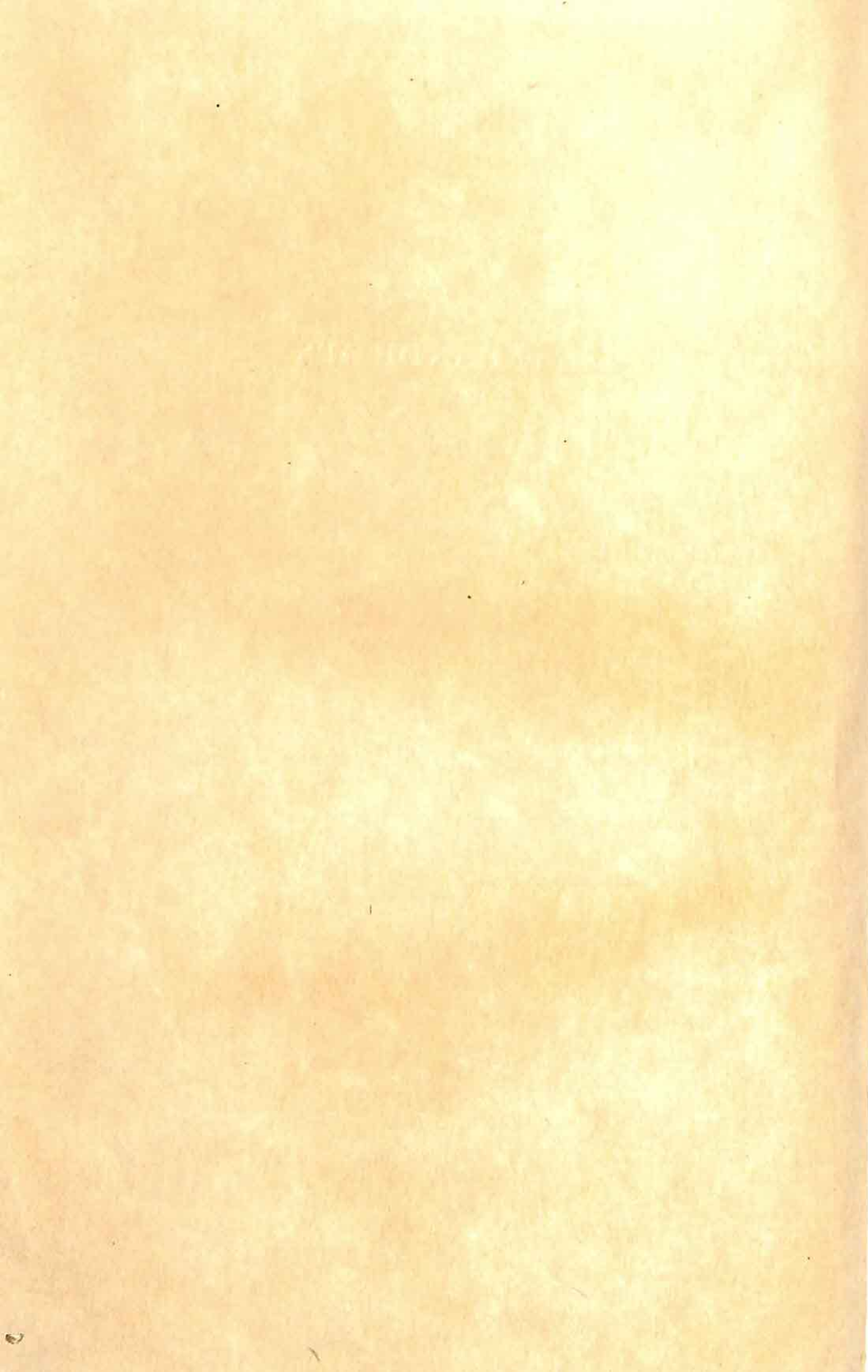


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CHILDREN

with

Mental and Physical HANDICAPS

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CHILDREN

with

Mental and Physical

HANDICAPS

by

J. E. WALLACE WALLIN, Ph.D.

Visiting Professor of Clinical Psychology, Upsala College. Formerly Director of Psycho-educational Clinics and Special Education and Mental Hygiene Departments at the University of Pittsburgh, and Miami University, and in the Departments of Education in St. Louis, Baltimore, Wilmington, and Delaware; Visiting Professor at Duke, Johns Hopkins, New York, Chicago, Virginia, California, and other Universities. Former President and Secretary of State Directors of Special Education



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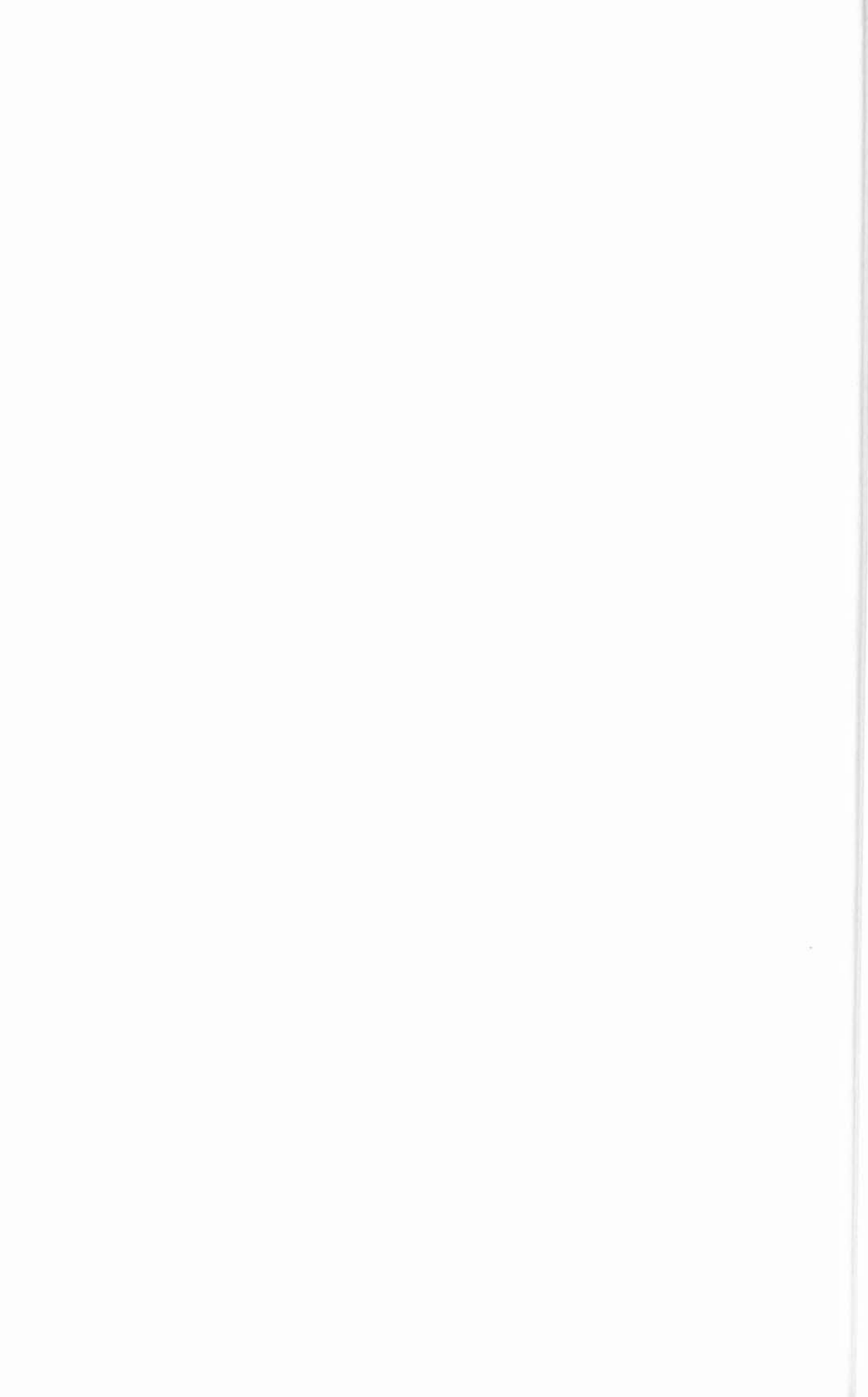
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To Barbara Ann & George Weatherby, III

*& All who are just embarking upon Life's
Greatest Adventure . . . who look hope-
fully to the Men of Science, Understand-
ing, & Goodwill to rid the world of the
twin curses of blighted brains & broken
bodies*



PREFACE

This book is the product of almost 40 years of first-hand experience as director, supervisor, and instructor in the fields of education and treatment of all kinds of handicapped children, clinical and abnormal psychology, and mental hygiene, and of extensive research in the technical writings, including recent publications. In a sense, it is an amplification of some of the material that appeared in the author's *Education of Handicapped Children*. Much the greater part of the volume consists of completely new material based on a quarter century of additional experience and surveys of the most recent findings of hundreds of investigators.

The book attempts to supply the need for an up-to-date reference book for the intelligent lay reader and, particularly, for the professional workers in the fields of special education, handicapped children, mental defect, clinical and abnormal psychology, child guidance, mental hygiene, social service, psychiatry, and pediatrics. It can also serve in the areas covered by advanced students in psychology, education, and medicine, and, more particularly, in special education, clinical psychology, mental and physical defect, orthopedics, and neuropsychiatry.

Lists of recent references, especially to American contributions, have been supplied for the benefit of those students and investigators who wish to do further reading on the different topics considered. Many of the citations contain additional references. Older references have, in some cases, been cited because of the influence the writers have exerted in shaping subsequent developments in general and the author's thinking in particular. Many of the older publica-

tions, which have been responsible in a large measure for dominant current trends, still have much to contribute.

It has not always been possible to obtain access to some of the originals cited in the text, even in out-of-state libraries that have been visited. It has therefore been necessary in a limited number of cases to utilize secondary sources, with the possibility of minor errors that such a procedure usually involves. This handicap accounts for the occasional divergence in the citation of references from the standard pattern adopted. The determination to supply the given names of all writers referred to, in order to indicate their sex, has at times been frustrated by the failure of the writers or bibliographies to furnish this item.

With the vigorous research carried on in the medical and biochemical laboratories during the last quarter century has come the knowledge that there are far more special types of mental defectiveness than was thought to be the case in the early part of the century. No book dealing with the description and classification of grades and types of mental defectives would be complete that did not devote at least a few paragraphs or pages to these more recently discovered rare types. Although space devoted to these types in this book is held to a minimum, ample references are supplied for those who desire fuller information.

It is a pleasure to express my appreciation to all those who have cooperated in the preparation of the book, including Dr. Elise H. Martens, Chief of the Division of Exceptional Children and Youth, U. S. Office of Education, and Dr. Samuel A. Kirk, Professor of Education (serving as head of the department for the education of handicapped children), College of Education, University of Illinois, who offered sundry suggestions after reading the original draft of the manuscript; Dr. Christine P. Ingram, Director of the Department of Special Education in the Rochester, New York, Public Schools, who offered suggestions on certain curricular matters; Godfrey D. Stevens, Instructor in the Division of Education

for Exceptional Children, Milwaukee State Teachers College, who read the incomplete draft and offered various comments and suggestions; Dr. Arthur A. Whitney, Superintendent of the Elwyn Training School, and Dr. Ralph H. Major, Professor of Internal Medicine, University of Kansas Medical Center, and Dr. Louis A. Lurie, Director of the Child Guidance Home, Cincinnati, Ohio, who supplied many of the photographs; and Dr. George A. Jervis, Dr. Hans Lowenbach, Dr. Robert W. Graves, and Dr. George J. Boines for critical reviews of various chapters. Other acknowledgments are expressed at appropriate points in the text. Mr. William Papanestor assisted in proofreading the volume. Miss Virginia Wallin assisted in the preparation of portions of the manuscript and in proofreading certain chapters.

J. E. WALLACE WALLIN

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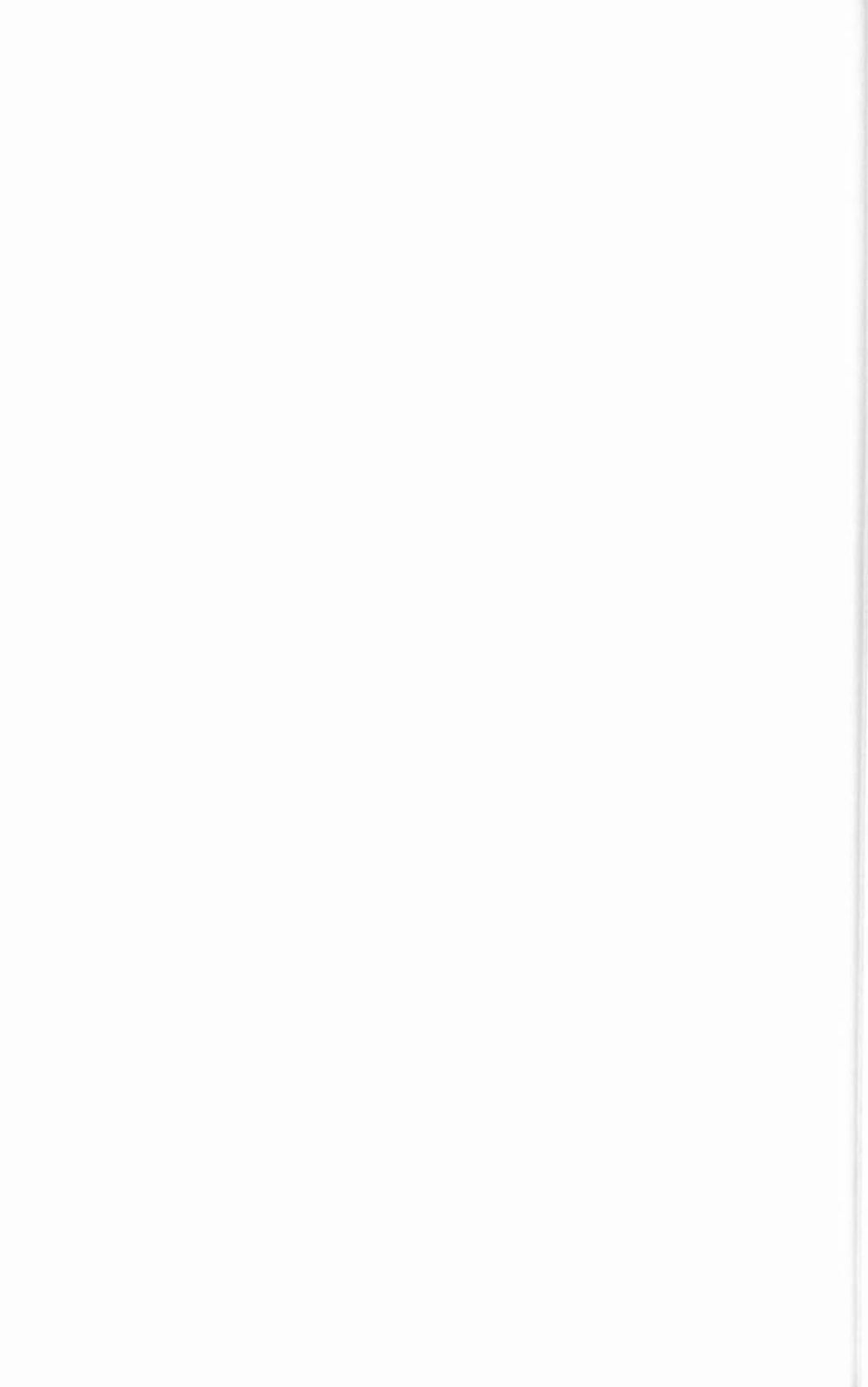
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CHILDREN

with

Mental and Physical HANDICAPS



Chapter 1

INTRODUCTION

Delimitation of the subject matter

Children are subject to many kinds of physical defects and anomalies and many kinds and degrees of psychological, educational, and social handicaps and maladjustments that interfere with their happy and successful adjustments to their physical environment and to the society in which they live. These multitudinous handicaps can be considered from many points of view—medical, psychological, mental hygiene, psychiatric, educational, or social. Detailed consideration has already been accorded a considerable variety of mental abnormalities, personality maladjustments, and physical deviations to which *homo sapiens* is subject from the psychological and mental hygiene standpoints in four other volumes.¹ Many of the problems discussed in these books can be given little or no consideration in the present volume, which is limited to the discussion of the problems of the physical (medical), psychological, educational, and social classification of mentally handicapped children, the causation of men-

¹ Intellectual, motor, emotional, and temperamental abnormalities and deviations, general and specific, have been treated in detail from the standpoint of the clinical psychologist in *Clinical and Abnormal Psychology*. Boston: Houghton Mifflin Company, 1928; and a great variety of personality maladjustments is considered from the mental hygiene point of view in *Personality Maladjustments and Mental Hygiene* (2d edition). New York: McGraw-Hill Book Company, Inc., 1949; *Minor Mental Maladjustments in Normal People*. Durham, N. C.: Duke University Press, 1946. *The Mental Health of the School Child*. New Haven: Yale University Press, 1914 and 1917, has long been out of print but is available in many libraries.

tal defects, and the clinical description, treatment, training, and education of the special clinical types.²

Public school teachers of mentally handicapped children and school administrators should, of course, be particularly conversant with all the problems presented by the large army of the simple, nonclinical kinds of mentally deficient and retarded children. A second volume to follow this will deal with the special educational problems of the group of mentally handicapped children who do not belong to any special clinical type. It will present a detailed overview of the problem of the public school education of the general run of mentally and educationally handicapped children of various levels of ability, whether permanently or temporarily disabled, whether their disablement is constitutional or acquired, and the social and eugenic problems they create in society.

Proportion of mental defectives among different clinical types

It is pertinent at this point to emphasize that not all of the members of the groups of children considered in this volume are mentally deficient or even backward. The ratio of mental defectives among the different types varies enormously. It varies from a maximum of almost 100 per cent for frank mongols, decided, untreated cretins, pronounced microcephalics, and various types associated with faulty metabolism of the nerve cells (infiltration with lipoids of fat); to a minimum of about one-third of the cerebrally palsied, perhaps not over 10 per cent of infantile paralysis cases, about 50 per

²The antecedents of the present text include, besides many brief articles, particularly the following: *Experimental Studies of Mental Defectives*. Baltimore: Warwick & York, Inc., 1912; *Problems of Subnormality*. Yonkers: World Book Company, 1917 and 1921 (a detailed treatment of the concept and criteria of feeble-mindedness and its implications); "The Achievement of Subnormal Children in Standardized Educational Tests," *Miami University Bulletin*, XX, 7, 1922; "Studies of Mental Defects and Handicaps," *Miami University Bulletin*, XXII, 5, 1924; *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1939. These publications include discussions of many issues in greater detail.

cent of institutional epileptics and 10 to 25 per cent of the noninstitutional cases, depending upon the type of case and the severity and duration of the seizures, and from 10 to 60 per cent of encephalitics, depending upon the age at onset and the area of the brain invaded by the disease organism (virus). These estimates would be increased somewhat for the last four groups if the dull normals were included. It is obvious, therefore, that several groups are given consideration, not because most of the members are mentally subnormal—the preponderance of them are normal, near normal, and bright—but because the minority who are subnormal create complicated and difficult educational, social, and vocational problems on account of their double handicaps. These doubly handicapped children are capable of considerable improvement if they are provided with appropriate medical, psychological, educational, and social treatment. Whether these children of limited mental endowment are registered in the regular grades or in special classes for the mentally handicapped or for the physically (orthopedically) handicapped, it is imperative that their teachers possess the minimum of knowledge and understanding of their mental and physical condition that will enable them to minister effectively to their individual needs. Moreover, many of those who are intellectually normal or only slightly retarded may be subject to various specific mental handicaps or personality maladjustments that spring from their primary disabilities which require discriminating psychological, educational, and mental hygiene treatment. All workers who serve mentally limited and physically handicapped children, whether teachers in the regular grades or in different kinds of special classes, medical specialists, psychologists, social workers, or educational or vocational counselors, should be conversant with the special problems these children create because of their limited mental potentialities. The noninclusion of the mentally subnormal speech defectives, visually and auditorially handicapped, and juvenile malbehavior cases or of-

fenders is due, in part, to the relatively recent appearance of comprehensive treatises in all these areas and, in part, to the necessity of limiting the size of this book.

Progressive types of mental deterioration contrasted with the ordinary primary ament

Attention should be called to an important distinction found in different kinds of mental defectives. The typical mentally deficient (and also retarded) child develops or improves mentally at a retarded pace to his maximum and then remains stationary for years until, if he lives long enough, dementia or deterioration slowly supervenes as a result of progressive senility. He does not dement or deteriorate during the period of growth and development, except as a result of some disease, or organic injury, or, possibly, extreme lack of environmental stimulation, or psychic traumas, or emotional blockages. Some of these slowdowns, stoppages, or regressions may represent temporary stagnations and may be pseudo-deteriorations rather than genuine. In certain types, however, marked irreversible mental deterioration may be, and often is, an outstanding characteristic of the syndrome. Among the types which may become increasingly deficient are most of the nerve-degeneration groups considered in Chapter 13, some juvenile epileptics with severe unchecked grand mal convulsions, many encephalitics, and many progressive juvenile hydrocephalics who do not respond to treatment. The victims of cerebral syphilis who do not respond to the newer treatments for neuro-syphilis represent forms of acquired brain disease that sometimes progress either to profound dementia or to mental alienation with gross mental deterioration.

Confusing terminology

There are many degrees and varieties of mental deficiency to which many terms have been applied in bygone years, often without much attempt at analysis or exact definition.

Among such terms may be mentioned: mental retardation, backwardness, dullness, slowness, stupidity, or impairment; lack of alertness; mental incapacity, inefficiency, incompetency, or inferiority; incomplete mental development; weakness of mind; subaverage mentality; subnormality; mental arrest; mental limitation; limited development, or arrested or delayed development; atypical mentality; mental deterioration or degeneration; dementia; amentia; mental defect, mental defectiveness, or mental deficiency; feeble-mindedness, hypophrenia, or oligophrenia; moronity, imbecility, and idiocy; the mentally limited, mentally handicapped children, or mentally underprivileged children. Some of these terms have been used in a highly specific or technical sense—amentia, imbecility, or idiocy; others in a very indefinite or general sense—retardation, backwardness, dullness, or subnormality. Some words have been used in a highly restricted sense by some writers and in a very general sense by others—the words “mental retardation” are used for a slight degree of mental defect or as an equivalent of mental defectiveness, and “subnormality” is used both as a generic concept or as an equivalent of feeble-mindedness. Some terms have been used as euphemistic synonyms for harsh-sounding words that supposedly carry unfortunate connotations, such as the use of mental retardation or arrest, or backwardness, or slow learning ability, which have been employed as substitutes for feeble-mindedness or subnormality. In Great Britain the term “feeble-mindedness” has been limited to the highest grade of mental defectives (called morons in the United States), and amentia has been used as an equivalent of mental defectiveness. In the United States feeble-mindedness and mental defectiveness and, to a limited extent, the more technical terms hypophrenia and oligophrenia have ordinarily been used as synonymous generic terms to include all grades of mental defectives—idiots, imbeciles, and morons. The more popular term “mental deficiency” has carried many connotations: it has been used as a mild synonym for men-

tal defectiveness or feeble-mindedness by Shuttleworth and Potts, Berry and Gordon, Tredgold, and many others: it has sometimes been restricted to the higher grades of mental defectiveness or to the borderline level of subnormality, and has also been arbitrarily defined in terms of minimum and maximum Binet I. Q.'s and intelligence levels and achievement levels to set standards for admission to public school special classes for children of the most limited levels of ability.³ Surely, "confusion doubly confounded" has reigned supreme for decades in the field of terminology as applied to children who deviate in the minus direction from the typical or modal child in the curve of distribution of human capacity. Furthermore, the confusion is enhanced by a plethora of other terms for various kinds of specific disabilities and personality anomalies often coexistent with generic mental subnormality. Such coexistences complicate the problems of diagnosis and classification.

The connotations of certain basic terms will be discussed in further detail in later chapters in the effort to attach a semblance of technical definiteness to these terms. In this book the terms *amentia*, mental defectiveness, and feeble-mindedness are employed to include all grades of mental defectiveness properly diagnosable as socially and economically inadequate because of *serious, permanent* intellectual defect or serious, permanent lack of capacity (the socio-legal connotation), and the terms "mentally handicapped children" and "subnormal children" are used in the generic sense to include all degrees of intellectual arrest, impairment, or retardation from pronounced degrees of mental defectiveness to merely slight amounts of backwardness. The designation "mentally handicapped" (and, to a lesser degree, the term "mentally subnormal") ordinarily does not carry the opprobrious implications that sometimes attach to the designations "feeble-minded," "mentally defective," or "mentally defi-

³ See Wallin, J. E. W., *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1924, p. 103.

cient." "Mentally handicapped children" constitutes an acceptable parallel to the large contrasting group of "physically handicapped children." Just as the term physically handicapped children includes all children who are beneath par physically through disease, injury, or developmental defect, so the term mentally handicapped will be used to include all children falling short of intellectual normality through disease, accident, or limited native endowment as well as children who manifest specific mental limitations or aberrations. The seriously mentally handicapped groups are proper subjects for diagnostic, remedial, or corrective instruction and mental hygiene treatment. This is also true of the physically handicapped groups.

Even children with slight intellectual defects often require special educational or mental hygiene treatment because of coincidental or overlapping disturbances or maladjustments. The child functions at all times as an organic whole, and deficiencies in the intellectual functions are often associated with emotional, temperamental, social, and educational involvements.

Children with Mental and Physical Handicaps has been chosen as the title of this book in preference to "Mentally Handicapped Children" because of the inclusion of several types of physically handicapped children, most of whom are not mentally defective. Many of these children create problems because of correlated or associated personality maladjustments or vocational limitations.

The material in this book is intended to supply not only a body of descriptive and explanatory facts but also the groundwork for the mastery of correlated educational, psychological, social, and medical problems in their practical implications. Although the student of the social sciences is mainly concerned with the practical social, educational, and psychological bearings of deviations from the normal, his information would be fragmentary and sophomoric, his counsel would

tend to be empirical, dilettantish, or fallacious, and his services might be indifferent, ineffectual, or harmful if he did not possess a grasp of at least the main outline of the medical phases of retarded and defective development, stripped of some of the minutiae that are of interest mainly to the medical investigator.

Order of treatment of the topics

The problems of classification and diagnosis and the relevant facts in connection therewith will be considered from the standpoint of psychological characteristics and criteria, of causation, of educability and educational characteristics, of socio-economic competency, and of clinical and physical types. In the opening chapters the contrasting theories and concepts of mental defectiveness will be compared, and definitions of mental defectiveness and backwardness will be formulated from five standpoints, based on certain essential and correlated criteria.

All of the chapters dealing with special and clinical types are actually in one main section, but are dealt with separately purely because of the length of coverage. The discussion of the causes of mental defectiveness in general and of causes, characteristics, and explanations of the different special types should supply desirable interpretative orientation and background for the consideration of the other phases of the problems.

All teachers of handicapped children will be more understanding, tolerant, and capable if they possess some knowledge regarding the nature and causation of the different special clinical (physical) types and the educational, social, and medical problems they present. They will have to handle some of these children in classes occasionally. These children are entitled to as much skilled instruction as any of the nonclinical kinds of mental, educational, and physical deviates.

In this field, as in any other field of specialized knowledge.

one cannot suffer from too much learning and insight. The danger lies in the opposite direction. There is much truth in Alexander Pope's adage for all who must wrestle with difficult problems: "Drink deep, or taste not the Pierian spring." The mere fact that some of the ramifications of some topics may be difficult does not constitute a legitimate reason for shirking or blinding one's self, ostrich-like, to their existence. However, an attempt will be made in the more technical sections to couch the presentation in terms that can be understood by the lay reader. It is, of course, very difficult to avoid the use of recognized technical terms in any scientific presentation, and also it is inadvisable to do so. After all, the correct use of technical terms is indispensable for the organization and control of thought and its correct expression in any area of learning. Thumb-nail definitions of the more technical terms will be supplied in parenthetical statements or footnotes to lighten the task.

Whenever it was possible the given name of the authors referred to in the text has been supplied on its first appearance. In many cases the initials of the writer only have been available. To aid the student in locating articles and books by various writers in their special fields, every effort has been made to identify the authors as far as possible when they are cited.

Because of the lack of uniformity in citing ages and mental ages, the decimal system is used when the original research reported findings with the decimal method. When, however, the months have not been turned into decimals, they are cited as 6-2, meaning six years and two months, not six and two-tenths years (6.2).

Chapter 2

MULTIPLE DEFINITIONS OF MENTAL DEFECTIVENESS

The confusion that exists in the terminology in current use, as pointed out in the Introduction, also applies to the concept of mental deficiency. Yepsen, from an analysis of "one hundred or more criteria and descriptions making up the definitions which have appeared in the literature," reached the conclusion that "none actually defines mental deficiency. All of the definitions are descriptive of the results of mental deficiency."¹ They were not based on the basic condition, but on symptoms and end-products of the condition—learning deficiency, inability to perceive meaningful relationships, social insufficiency, economic dependency, lack of adaptability or resiliency, and the like. In spite of a century and a half of prolonged scientific research in the field of mental subnormality, definitions will doubtless continue to a large extent on the symptomatic and descriptive level until the gaps in our understanding of the nature and causes of various kinds and degrees of amentia have been removed. Descriptive and symptomatic definitions, however, in spite of their limitations, constitute an essential aspect of a differential diagnosis.

Definitions of mental defectiveness

The definition of mental defectiveness will vary with the point of view. Mental defectiveness can be defined from at

¹Yepsen, Lloyd N., "Defining Mental Deficiency," *American Journal of Mental Deficiency*, 1911, 46: 200-205.

least five standpoints which, although confessedly not of equal importance, are germane to the problem. Each aspect adds an important facet to the total picture. It will serve our purpose best to begin with the etiological backgrounds, anatomical and psychological, of the concept.

A. *Anatomical definition.* Anatomically mental defectiveness represents a condition of serious and permanent nervous defect, due to arrested neural development (agenesis), or damaged nervous tissue, affecting particularly the brain and especially the external layer of gray cells (the cortex), and dating from the prenatal or early postnatal period of the child's development. The terminus of the postnatal period has not been definitely determined. Woodrow² advanced the theory in the second decade of this century that mental defectiveness cannot be acquired after the age of five, at which time the brain is said to have attained about 90 per cent of its final weight. The attempt to reach such a conclusion from a consideration of brain weight alone may, however, be fallacious, for it may be precisely the higher functions, cerebral and psychic, which mature after the age of five, say between five and nine, which may be most seriously impaired in mental defectiveness. Should these functions normally attain a stage of development by the age of ten, say, that will enable the individual to lead an independent existence, then serious brain injuries (lesions) occurring any time during the first nine years might produce mental defectiveness, in the technical sense of the term. In 1927, however, the British Mental Deficiency Act advanced the age limit to eighteen.

According to this concept, the essential basis of mental defectiveness is defective morphology of the nerve cells of the brain (cerebral neurones), which is severe and irreparable, whether due to the biological tendency toward variation, or to lack of potentiality for maturation (hypoplasia), or to

²"It is a remarkable fact that no child ever becomes feeble-minded after the age of four or five." Woodrow, Herbert, *Brightness and Dullness in Children*. Philadelphia: J. B. Lippincott Company, 1919, p. 69.

some disease or injury (pathological basis). The condition, however, is often associated with various accessory or inconstant somatic defects or diseases, such as defects of metabolism, circulation, endocrine functioning, sensory acuity, or neuromuscular efficiency. The mentally defective as a class are somewhat underdeveloped in strength, muscular tonicity, weight, and particularly height. It is generally held that they are more susceptible to contagious diseases and that they suffer more frequently from the ordinary physical defects and from glandular, cardiac, and circulatory defects. The record of past diseases for 872 consecutive cases examined in the St. Louis psychoeducational clinic years ago gave an average of 2.8 for those diagnosed as mentally defective, compared with 2.5 for those not mentally defective. Some diseases were far more prevalent among the mental defectives: cerebrospinal meningitis, by 345 per cent; infantile paralysis, by 322 per cent; and epileptic convulsions by 83 per cent.³

The primary (hereditary) type of mental defectiveness is a static defective neural condition and not an active disease process curable by medication. In some of the special types, for instance, hydrocephalus, active syphilis, epilepsy, cretinism, and encephalitis, the condition may be due to an active pathological process which is amenable to improvement or cure from medical or surgical treatment. Also the putative cerebral defect does not afford a reliable basis for the diagnosis of mental deficiency or for prognosis, because the exact brain condition cannot be determined in the large majority of cases during the life of the individual. Present techniques of post mortem brain examinations may or may not reveal the etiological basis for the child's abnormality. In the case of acquired cases attributable to cerebral lesions.

³ For details see Wallin, J. E. W., "Causative Factors of Mental Inferiority and the Prevention of Degeneracy," *Proceedings of the Forty-sixth Annual Session of the American Association for the Study of the Feeble-minded, Journal of Psycho-Asthenics*, 1922, 27: 75-116. For a résumé of discrepancies in the data on the physical status of subnormal and normal children see Wallin, J. E. W., *Clinical and Abnormal Psychology*. Boston: Houghton Mifflin Company, 1928, pp. 54-70.

surgical procedures might bring mental improvement although the prognosis in such cases is usually unfavorable. However basic the organic cerebral background may be for the definition of mental defectiveness, the anatomical criterion is not of great value in the practical diagnosis of the condition and in the differentiation of different levels of subnormality.

B. *Psychological definition.* Psychologically, mental defectiveness represents a condition of mental nondevelopment (agenesis), arrest, deficiency, or deterioration which is very grave and permanent, which dates from early life, and which always affects the intelligence, judgment, or understanding and the capacity for social and economic adjustment (the socio-legal connotation of the term), and which may also and usually does to some extent affect the emotional, instinctive, motor, volitional, and moral traits of the individual. The intelligence defect is a basic and constant feature; without a radical defect in the sphere of intelligence the child could not be considered mentally defective no matter how ignorant or illiterate he might be, or how abnormal in his emotional, instinctive, volitional, or moral reactions.

Three distinct psychological criteria are postulated in this definition.

1. **EARLY ONSET.** The defect must date from the child's intrauterine or early postnatal life. It must have occurred prior to the maturation of the mental functions, how long prior has not been determined. Some hold that the arrest or defect must have taken place during the first four or five years of life, but our knowledge is too imperfect to permit a categorical pronouncement on this point. The "upping" of the limit to eighteen years by British Statute, apparently for the purpose of dealing with older deteriorating cases of encephalitis lethargica as certifiable mental defectives, is of dubious propriety. Such cases would, perhaps, be more accurately classified as demented rather than as amented. Traditionally, mental defectiveness, in the accepted narrow tech-

nical connotation of the term, means amentia—nondevelopment of mind, as distinct from dementia—loss or decay of mind. The mentally defective has never had more mind before, unless dementia has been superimposed upon a background of amentia. He has never had normal potentials, unless, indeed, cerebral degeneration of an originally sound brain began in early life, in which case the child would still be regarded as an ament and not a dement. The dements, on the other hand, may have had normal potentials, and may have reached normal mental maturity—the brain cells may have been sound originally, but they have degenerated. Many dements deteriorate to the level of aments and may possess about the same ability as an ament of the same intelligence level. Nevertheless, a dement should not be confused with an ament. There is ordinarily a distinct difference between a dement and an ament even when the intelligence level is the same. Several differences between the two may be noted.

(a) The ament is usually good-natured, carefree, and confident. The happy-go-lucky disposition of the typical ament (exceptions exist, of course) is fittingly expressed in the quatrain of the college wag:

See the happy moron,
He doesn't give a damn;
I wish I were a moron,
Who knows, perhaps I am.

The dement, on the other hand, tends to be sullen and heavy. He often loses his capacity to feel, whereas the ament's capacity to feel or react to his feelings is not lost, although it may be temporarily diminished, exalted, or modified.

(b) Some investigations show that at least some types of dements (or psychotics) are more uneven or irregular than aments in their mental make-up as determined by scatter on the Binet scale. Our early results showed that parietic and

senile dementers scattered more over the Binet scale than mental defectives of various grades. The Presseys found that the total irregularity for the 20 tests in the Yerkes-Bridges point scale, based on the scores for normal children of the same intelligence age, was 20 points for dementia praecox cases in the early stages, 22 points for deteriorated chronic alcoholics, as compared with 17 points for feeble-minded cases of the same intelligence ages. Based on five more highly differential tests (definitions of concrete and abstract words, absurdities, and disarranged sentences), the corresponding differences were 6.9, 8.2, and 4.7. Curtis and also Wells and Kelley found that the scatter was slightly greater for paretics than for praecox cases, and slightly greater for the praecox than for the manic-depressive cases, not all of whom, however, were deteriorated.⁴

(c) Mental defectives have never shown much initiative, adaptability to changing conditions, resourcefulness, imagination, foresight, planning ability, discretion, reasoning ability, or motivation (their mental make-up is one of rigidity), whereas the dementers may have functioned normally in these respects in their pre-psychotic careers.

(d) Wrinkles frequently tend to furrow the brow of the dement, but the forehead of the ament may remain smooth until a late age, especially if he is of low intelligence.

(2) IRREMEDIABILITY. A diagnosis of genuine mental defectiveness has always been predicated on the assumption that the defect is incurable—the diagnosis of mental defectiveness involves a prognosis of incurability. The restoration of a

⁴Curtis, Josephine W., "Point Scale Examinations on the High Grade Feeble-Minded and Insane," *Journal of Abnormal Psychology*, 1918, 77-118.

Pressey, Sidney L., "Irregularity in a Mental Examination as a Measure of Its Reliability," *Psychological Clinic*, 1919, 236 f.; "Irregularity on a Psychological Examination as a Measure of Mental Deterioration," *Journal of Abnormal Psychology*, 1917, 3 f.; "A Comparison of a Girls' Reform School, Attendants at a State Hospital for the Insane, and Public School Children by Means of Certain Tests of Intelligence," *Journal of Criminal Law and Criminology*, 1921, 2: 258 f.

Wells, Frederic L., and Kelley, C. M., "Intelligence and Psychosis," *American Journal of Insanity*, 1920, 17-45.

deficient to normality demonstrates that he was not mentally defective, but only temporarily retarded. There may be certain exceptions to this broad statement. If thyroid extract is administered early enough, it may be possible to raise to mental normality some cretins whom we should be obliged to consider genuinely mentally defective before treatment and who would have continued as such without treatment. It may also be possible to restore some active syphilitics, hydrocephalics, encephalitics, and persons whose mental functioning is inhibited or disturbed by seizures or by emotional blockages. Possibly the pseudomental defectiveness of some potentially normal victims of environmental deprivation may be overcome through "environmental stimulation" in accordance with the Iowa postulate, discussed on page 135. An attitude of flippant dogmatism on this point should be replaced by the recognition of its possibility in a limited number of cases. Frankly, the writer has never, in almost 40 years of clinical practice or supervision of special class work, known a genuine mentally defective child of the primary type who was indubitably restored to complete normality, although many have been considerably improved. The diagnosis of these cases has, of course, been based on the synoptic findings of multiphase clinical examinations and not on mere test findings or I. Q.'s.

But many grades of mental subnormality exist that are not remediable, although they do not constitute mental defectiveness. Hence, a third qualification must be added.

(3) GRAVITY OF DEFICIENCY. The intelligence deficiency must be very serious. Exactly how serious must the defect be to constitute feeble-mindedness? On the supposition that there can be no mental defectiveness without a serious deficiency of intelligence, various attempts have been made to formulate precise standards in terms of the amount of intelligence defect.

(a) *Absolute amount of retardation in years.* Goddard, in 1910, proposed to call a child feeble-minded if "more than

two years backward" by the 1908 Binet scale when "under nine years of age," and if "more than three years backward" when "above nine," subsequently modifying the standard so as to provide that a child who is four years or more backward in intelligence is "without doubt feeble-minded."⁵ Doll modified the formula slightly, "Feeble-mindedness is defined psychologically as intellectual retardation of two years at an age below nine or three years at an age above nine." No experimental evidence was offered in proof of the correctness of these standards, but they hark back to a tentative basis for the preliminary selection of candidates for special classes suggested by Binet and Simon: "A retardation of three years indicates a child who should be regarded as suspect." "According to a convention accepted in Belgium, which we modify slightly, the retardation which determines a child as a defective is two years when the child is under nine, and three years when he is past his ninth birthday." Binet and Simon, however, took pains to point out that "these distinctions are pedagogical," and only permit "the making of a first selection . . . without being final."

CRITICISM. In this country thousands of children were for years assigned to special classes or committed to state residential institutions on the basis of these absolute standards of defectiveness. These early absolute standards have been almost wholly abandoned now, however, because they relegated so many children to the mental defectiveness category who could not be so diagnosed on the basis of the more decisive social criterion and because the significance of a year of intelligence retardation varies with the etiological background and the complications accompanying each case and especially with the chronological age of the child. Scientific measurements must be based on a unit of measurement that remains invariable. But a year of difference in intelligence is an

⁵ Goddard, Henry H., "The Binet Measuring Scale of Intelligence, What It Is and How It Is To Be Used," *Training School Bulletin*, 1914, 86-91; "The Binet-Simon Measuring Scale for Intelligence," *Training School Bulletin*, January, 1910, 3.

inconstant, not a constant, unit of measurement. It does not have the same significance in the different levels of the scale. One year of difference in early life is much greater than one year of difference in later childhood or adolescence. One year of retardation in a two-year-old child is appreciably greater than one year of retardation in an eight-year-old or in a fifteen-year-old. For a two-year-old, a year of retardation, if reliably obtained, constitutes a grave defect; for a fifteen-year-old, it is barely noticeable.⁶ To overcome this inherent defect in the absolute year unit, a relative or fractional standard was suggested as early as 1910, which Stern subsequently popularized under the designation "Intelligence Quotient" (I. Q.).⁷

(b) *The Intelligence Quotient, Intelligence Ratio (I. R.), or Mental Ratio.* The I. Q. is obtained by dividing the chronological age into the intelligence age as determined by the Binet-Simon or any other "test of intelligence," whether individual or group, or whether verbal, linguistic, mechanical, or psychomotor. An eight-year-old child with a test age of six has an I. Q. of 75, which indicates that he possesses 75 per cent of normal intelligence as determined by the test scale.

Because of its simplicity and reputed value, the practice of diagnosing mental defectiveness or feeble-mindedness by means of the Binet I. Q. became well-nigh universal after the appearance of the Stanford revision of the Binet scale in 1916. Stern, from merely computing the quotients for a group of auxiliary school pupils in Germany, tested and classified by someone else, suggested these diagnostic criteria: "not abnormal," 81 to 90; morons, 71 to 80; imbeciles, 61 to 70. Morons, according to Stern, possess "three-quarters intelli-

⁶ It would lead us too far afield to enter into a discussion of other limitations of the mental age concept. Cf. Wechsler, David, *Measurement of Adult Intelligence* (2d edition). Baltimore: William Wood & Company, 1941, pp. 20 f., 41 f.

⁷ Stern, William, *The Psychological Methods of Testing Intelligence*. Baltimore: Warwick & York, Inc., 1914.

gence," and imbeciles a "scant two-thirds intelligence." Terman arbitrarily adopted a range of 10, 20, or 30 I. Q.'s for each category, and proposed the following I. Q. standards of diagnosis, based on the Stanford-Binet scale (but not derived from the actual clinical diagnosis of the children examined) :

Above 140,	"near" genius or genius
120-140,	very superior intelligence
110-120,	superior intelligence
90-110,	normal or average intelligence
80-90,	dullness, rarely classifiable as feeble-mindedness
70-80,	borderline deficiency, sometimes classifiable as dullness, often as feeble-mindedness
Below 70,	definite feeble-mindedness
50-70,	morons
20 or 25-50,	imbeciles
Below 20 or 25,	idiots ⁸

Burt "provisionally fixed . . . the line of demarcation . . . between normals and defectives" at an I. Q. of 70 for children. For adults he apparently drew the line at 50. Below 50, he says, the case is an "institutional" one, and between 50 and 60 a "supervision" case.⁹ Twelve years later Pintner placed the upper limit of feeble-mindedness "somewhere in the neighborhood of I. Q. 60,"¹⁰ and 17 years later, on the basis of the Terman-Merrill 1937 Stanford-Binet, Merrill drew the line at I. Q. 70,¹¹ and Bernreuter and Carr¹² drew it at I. Q. 60.

⁸ Terman, Lewis M., *The Measurement of Intelligence*. Boston: Houghton Mifflin Company, 1916, p. 79.

⁹ Burt, Cyril L., *Mental and Scholastic Tests*. London: P. S. King and Son, 1921, pp. 168, 188.

¹⁰ Pintner, Rudolf, "The Feeble-minded Child," in *Handbook of Child Psychology* (2d edition), Murchison, Carl (editor). Worcester: Clark University Press, 1933, pp. 802-857. Of 17,502 school children selected at random, 1.28 per cent fell below I. Q. 60 and 5.72 per cent below I. Q. 70.

¹¹ Merrill, Maud A., "Significance of the I. Q.'s on the Revised Stanford Binet Scales," *Journal of Educational Psychology*, December, 1938, 641-651.

¹² Bernreuter, Robert G., and Carr, Edward J., "The Interpretation of I. Q.'s on the L-M Stanford-Binet," *Journal of Educational Psychology*, April, 1938, 312-314.

In 1941 Wechsler placed the mentally defective group at a distance of minus 3 P. E. or more from the mean, probably on the basis of the Bellevue-Wechsler scale, which would be equivalent to a maximum I. Q. of 65.¹³ His more recent classification, based on the "statistical concept of intelligence," is as follows:

<i>Classification</i>	<i>Statistical Limits in Terms of P. E.</i>	<i>Actual I. Q. Limits (Ages 10-60)</i>	<i>Per Cent Included</i>
Defective	-3 P. E. and below	65 and below	2.2
Borderline	-2 P. E. to -3 P. E.	66-69	6.7
Dull Normal	-1 P. E. to -2 P. E.	80-90	16.1
Average	-1 P. E. to +1 P. E.	91-110	50.0
Bright Normal	+1 P. E. to +2 P. E.	111-119	16.1
Superior	+2 P. E. to +3 P. E.	120-127	6.7
Very Superior	+3 P. E. and over	128 and over	2.2

The percentage distribution is based on the I. Q.'s.

The same year Porteus fixed the upper threshold of mental defectiveness at an I. Q. of 60, or an intelligence age of eight years and five months, based on the average I. Q. by the Binet and Porteus maze tests, "or other standard performance scale"; or an I. Q. of 65 or an intelligence age of nine years when these results are based on both the Binet and the maze tests.¹⁴ The author recognizes, however, that these standards would include some "borderline defective" cases.¹⁵

Table I shows the I. Q. values of the author's intelligence

¹³ Wechsler, *Measurement of Adult Intelligence* (2d edition), 1941, pp. 36 ff.

¹⁴ Porteus, Stanley D., *The Practice of Clinical Psychology*. New York: American Book Company, 1911, p. 247.

¹⁵ Some of the pertinent criticisms made by Wechsler throughout his book and by Porteus in his chapter on the feeble-minded child (pp. 228-255) of the diagnosis of mental defectiveness by rule of thumb on the basis of intelligence age or I. Q. were repeatedly voiced by the author, as an outgrowth of clinical experience, in numerous articles and books during the second decade of the century (e.g., in *The Mental Health of the School Child*. New Haven: Yale University Press, 1914, pp. 196 ff.; *Problems of Subnormality*. Yonkers: World Book Company, 1917, pp. 110-277). It may be stated, as a matter of historical record, that these criticisms for the most part fell on deaf ears, so convinced were some workers that all that was needed for the diagnosis of feeble-mindedness was a Binet test. The criticisms for some years were honored by their breach rather than by their observance.

categories or classifications for 411 St. Louis school children to whom the 1916 Stanford-Binet was administered during the school year 1917-18. All the tests, case summaries, and synoptic diagnoses were made by the writer on the basis of all the facts gathered in the case histories. The fields of inquiry covered in the histories included a special general physical examination (made by the school physicians), the personal and family histories (gathered by the school nurses), the detailed school record and personality evaluation (contributed by the teachers), and the results of earlier standardized tests.¹⁶ The initial data submitted on prepared standardized forms were supplemented by personal interviews, especially with the parents. The most striking thing revealed by a perusal of the table is the very wide range of extreme I. Q.'s in most of the categories and the extensive overlapping between the categories, because the clinical diagnoses are based on all the clinical data available on each case and not solely on the I. Q.¹⁷

CRITICISM. Because the I. Q. rapidly became a sort of fetish, it was necessary to sound frequent warning notes in the early days against the exaggerated importance attributed to the device. The I. Q. possesses unquestioned value within its natural limits of usefulness when it is accompanied by the indispensable data needed for the judicious interpretation of test findings, such as the examinee's chronological age, test age, objective and subjective factors at the time of the exami-

¹⁶ For the writer's "schema for the clinical study of mentally and educationally unusual children," see Wallin, *The Mental Health of the School Child*, pp. 429-446. Copies of the blanks in continuous use during many years are found in *Procedures for the Reporting of Handicapped Children, for Psychoeducational and Audiometric Examinations, and for the Establishment of Special Classes*, Department of Public Instruction, State of Delaware, 1942, pp. 49 ff. These examination schedules antedated Fernald's ten "fields of inquiry," referred to by one writer as the "prototype of all subsequent proposals," by several years: Fernald, Walter E., "Standardized Fields of Inquiry for Clinical Studies of Borderline Defectives," *Mental Hygiene*, 1917, 1:211-231.

¹⁷ Wallin, "The Value of the Intelligence Quotient for Individual Diagnosis," *Journal of Delinquency*, May, 1919, 118; *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1924, p. 68.

nation, especially the general psychosomatic condition¹⁸ and the emotional state, which may have influenced the subject's reaction pattern and which must be considered in the attempt to appraise the correctness of the obtained score, and the

TABLE I

Distribution of I. Q.'s in Each Clinical Classification, Based on Synoptic Findings on Referrals to a Public School Psychoeducational Clinic

Category	Average I. Q.	Median I. Q.	Modal I. Q.	Extreme I. Q. Range	Range Exclusive of 25% of the Extreme I. Q.'s
Normal	99	96		95 to 1.08	
Retarded	90	92		80 to 97	86 to 94
Backward	80	81	82	59 to 94	74 to 87
Borderline	72		75	56 to 84	66 to 80
Potential feeble-minded	66	67	67	50 to 79	61 to 71
Borderline and potential feeble-minded	72	70		56 to 84	64 to 77
Deferred	71	75		39 to 95	55 to 92
Morons	60	61	65	48 to 70	53 to 66
Potential morons	58	59		43 to 69	51 to 65
Imbeciles	46	47.5	56	21 to 65	31 to 58
Imbeciles and potential morons	52	54		21 to 69	40 to 62
Idiots	17			17	

genetic, psychological, social, educational, physical, and constitutional background of the deviations. The warnings issued in the past are still in need of emphasis. Although the I. Q. possesses undoubted value, especially when clinically derived, as the most practical single measure of relative brightness available, it is subject to a number of limitations

¹⁸ Fay, in emphasizing that "dullness and drowsiness" may follow a shift in the blood volume in the cranium, maintains that some examinees should be prepared for a psychological examination just as patients are now prepared for a basal metabolism by control of activities, diet, or rest. "The *post-traumatic* and *organic* types of mental retardation improve slightly but definitely by spinal drainage, if followed by strict limitation of fluid intake and dietary balance." Fay, Temple, "Basic Requirements for the Testing of Mental Acuity with Especial Reference to the Intracranial Pressure and Volume Relationships," *American Journal of Mental Deficiency*, January, 1947, 369-371.

that should be clearly recognized by those who make any practical use of I. Q. findings. Only a few of these can be mentioned here.¹⁹

(1) No particular I. Q. constitutes indubitable proof of mental defectiveness or feeble-mindedness in the socio-legal sense, except in perfectly obvious cases. The wide divergence in the proposed standards for diagnosing high grade feeble-mindedness, from a top I. Q. of 60 to 80 is presumptive evidence in favor of this assertion. The proposed I. Q. standard of 70 in the case of children, unfortunately, pertinaciously gripped the imagination of Binet testers for years. Thousands of children were committed to colonies as feeble-minded on this standard in spite of the early criticisms voiced against excessive reliance upon I. Q. diagnoses. The fact is that follow-up investigations of many ex-pupils from public school special classes have shown conclusively that many children who fall below Binet I. Q. 70 cannot be diagnosed as feeble-minded from the standpoint of socio-industrial competency.

There is little objection to adopting arbitrary I. Q. standards for the different grades of mentally subnormal children (backward cases, borderline cases, morons, imbeciles, and idiots) nor is it of much moment where the lines between these groups are drawn, provided it is clearly recognized that the divisions are arbitrary and that the terms are used in a purely intellectual sense for purposes of better classification or grouping—that is, if the term feeble-mindedness is given a purely intellectual connotation. But the cavalier transmutation of the I. Q. standards of diagnosis based on verbal tests of intelligence (and even performance or mechanical ability tests), however valuable they may be, into categories of social defectiveness is a wholly different matter fraught with grave social implications and is based on assumptions difficult to justify, at least so far as concerns the higher grades of defi-

¹⁹ For other comments on the I. Q. see Wallin, *Clinical and Abnormal Psychology*. Boston: Houghton Mifflin Company, 1928, pp. 201-208.

cients—those who range along the upper threshold of feeble-mindedness.

(2) Prognoses based on the I. Q.'s of young children are predicated on the assumption that the I. Q.'s will remain constant for normal persons during the periods of growth and maturity until deterioration sets in and that for mental defectives I. Q.'s will gradually decline or at least remain stationary during the period of maturation.²⁰ Although the Binet I. Q. does not change more than five or six points on the average, some children make unexpected gains whereas others suffer unexpected declines. We know from numerous retest investigations that the fluctuations in individual cases may be very large, possibly 20 to 30 points or even more. Unfortunately no reliable method has been devised which enables the examiner to determine in advance which examinees are subject to excessive variations and the direction that the changes will take. It is obvious, therefore, that prognoses based on I. Q.'s must necessarily be tentative, and must be expressed in very guarded terms. This is particularly true of I. Q.'s obtained from group tests.

(3) The I. Q.'s cannot be used reliably with adolescents (or very young children either) without the use of a corrective or attenuation formula to overcome the slackening in mental growth with increasing age. The author emphasized this requirement just as soon as Stern's proposal was applied

²⁰ This rule is not without exceptions. In the most recent study of retests of "institutionalized defectives" by "one of the revisions of the Binet test" made by various examiners at a mean interval of about four years, 62 per cent showed I. Q. losses, with a mean loss of 6.7 points; 31 per cent gains, with a mean gain of 4.7 points; and 8 per cent remained stationary, which, apparently, means that they had exactly the same I. Q.'s in successive testings. Apparently, the comparison of the successive I. Q.'s was based on different scales, in some cases at least. Since some patients had I. Q.'s up to 100 it is probable that many of the patients were not "mental defectives." See Sloan, William, and Harman, Harry H., "Constancy of the IQ in Mental Defectives," *Journal of Genetic Psychology*, December, 1947, 71: 177-185. See also Pintner, Rudolf, *Intelligence Testing*. New York: Henry Holt & Company, Inc., 1931, pp. 344 f.

for purposes of practical diagnosis.²¹ Various proposals have since been made to overcome the distorting influence of the chronological age factor, as we phrased it in our early criticism. Heinis²² proposed to divide the intelligence age by the chronological age after each had been translated into "growth curves." Terman and Merrill, in calculating the I. Q. for ages thirteen to sixteen, "cumulatively drop one out of every three additional months of chronological age and all of it after sixteen."²³ Wechsler substitutes for the usual formula,

$$\text{I. Q.} = \frac{\text{M. A. the formula}}{\text{C. A.}}$$

$$\text{I. Q.} = \frac{\text{Actual score obtained in a given test by the subject}}{\text{Expected mean score for age}}$$

The figure used in the denominator is the score made by an average person of the child's life age. The I. Q.'s are calculated from a base of -1 P. E. (probable error) from the mean, which gives -1 P. E. the value of I. Q. 90. The average, which contains 50 per cent of the cases, falls between $+1$ P. E. and -1 P. E. It is maintained that an I. Q. thus derived remains constant through life.²⁴ Use has also been made of percentile scores, standard scores, C scores, T scores, and F scores for obtaining constant measures.

(4) After all, the I. Q. is an x to be interpreted in the light of all the circumstances affecting each case, therefore different diagnoses will sometimes be made of individuals who have exactly the same I. Q. Because of the differences in the

²¹ See, e.g., Wallin, "Who Is Feeble-Minded? A Reply to Mr. Kohs," *Journal of Criminal Law and Criminology*, May, 1916, 67; "The Value of the Intelligence Quotient for Individual Diagnosis," *Journal of Delinquency*, May, 1919, 121 f.

²² Heinis, H. A., "Personal Constant," *Journal of Educational Psychology*, 1926, 163-186.

²³ Terman, Lewis M., and Merrill, Maud A., *Measuring Intelligence*. Boston: Houghton Mifflin Company, 1937, p. 30.

²⁴ Wechsler, *The Measurement of Adult Intelligence*, 1941, pp. 24-34.

attendant complications, the different intelligence categories will contain a considerable range of I. Q.'s and much overlapping will inevitably occur between adjacent I. Q. groups, as shown in Table I. The extent to which the diagnoses may differ can be shown by two extreme cases examined on the same day, each with an I. Q. of 66. One was a boy of 9.6 years, who had a Binet age of 6.4 and was diagnosed as a potential imbecile on the basis of the case history (anamnesis) and all the facts discovered at the time of the examination. He was an epileptic subject to severe seizures (for which the medical treatment at the time was largely futile) whose family history contained evidence of much neuroticism. Follow-up investigation for several years showed that he had little potentiality for development and stagnated as an imbecile. The other child, age 15.3, with a Binet age of 10.2, was diagnosed as "borderline or backward." Two years later he was earning \$17 a week, a good wage at the time, and on the basis of proved economic competency could not be diagnosed as lower than backward or dull normal.

It may be freely admitted that the Binet I. Q. validly obtained as a result of competent test administration is a substantial aid to diagnosis—I would not want to attempt a diagnosis involving deviation in ability level without it or an equivalent—but it should not be overworked.

(c) *The maximum intelligence-age level.* In 1910 the proposal was made to consider all older adolescents and adults as feeble-minded if they were unable to go beyond age twelve in the 1908 Binet-Simon scale. This proposal, although merely tentatively accepted by the American Association for the Study of the Feeble-Minded (now called The American Association on Mental Deficiency), was adopted categorically without evidence by a large number of American mental examiners until the data on the average intelligence-age level of the American soldiers became available in 1919. In point of fact, the evidence in support of the twelve-year standard at the time of its adoption was wholly inadequate. It was based

on about 400 institutional cases in the Vineland Training School. Of these only 3.7 per cent had a ten-year Binet age, only 1.3 per cent an eleven-year Binet age, only 1.8 per cent a twelve-year Binet age, and none thirteen. These data were based on the 1908 Binet scale which had no tests above age thirteen, only three tests in thirteen, only four in twelve, and only five in eleven. Moreover, as was proved later, most of the tests in the upper ages were too difficult. Obviously standards established on a handful of institutional cases by means of an imperfect scale with standards too difficult in the critical ages cannot be applied indiscriminately to the general run of the noninstitutional population.

On the basis of clinical studies, the writer, as early as 1912, was compelled to reject the classical twelve-year standard and tentatively fixed the upper level of the highest grades of mental defectives between nine and ten. Definite announcement of this conclusion was made in a paper on "Who Is Feeble-Minded?"²⁵ read before the American Psychological Association in December, 1915. Later investigation revealed that Binet and Simon had drawn the limit at practically the same level. Binet held, as reported by Stern, that the "moron" does not progress beyond the intelligence age of nine. Simon in 1915 wrote: "Provisionally it might be proposed to fix at nine years the upper level of mental debility." Later, Burt proposed the "mental age of eight," based on the Stanford-Binet, as the line of demarcation between adult "normals and defectives," which he apparently considers to be equivalent to "eight and a half or nine, according to earlier allocations" of the tests. Pintner accepted the standard of 8.6 adopted by the British Mental Deficiency Report of 1929, and Bernreuter and Carr offered the following 1937 Stanford-Binet age limits: borderline, 9 to 10.49; morons, 6 to 8.99; imbeciles, 3 to 5.99; and idiots, 0 to 2.99.

²⁵ Wallin, "Who Is Feeble-Minded?" *Journal of Criminal Law and Criminology*, January, 1916, 706-716; May, 1916, 56-78; July, 1916, 219-226. *Problems of Subnormality*, pp. 110-277.

EVIDENCE FROM THE EXAMINATION OF WORLD WAR I SOLDIERS. The psychological examination data for the inductees in World War II have not yet been made available,²⁶ but the results from the intelligence examination of a million and three-quarters of draftees in the United States Army of World War I should have discredited, once and for all, the twelve-year standard of feeble-mindedness. The army results showed that the average intelligence age of the American soldiers, based on the transmutation of the Alpha scores into Stanford-Binet ages, was between thirteen and fourteen for the white men and about ten for the colored. Of the white draft, 47 per cent had an intelligence age of twelve and less, 30 per cent of eleven and less, 18 per cent of ten and less, 10 per cent of nine and less, and 5 per cent of eight and less. The corresponding figures for the colored soldiers were 89, 79, 64, 48, and 32 per cent. These figures bear out the conclusion reached independently of the army results and long before they were available, that the possession of a ten-year mentality constitutes a strong presumption that the individual is not feeble-minded (not a moron, although a dullard), however abnormal or anomalous he may be in other respects. Clinical studies during the last two decades have demonstrated that many stable, industrious persons exist who do not reach a nine-year level by the verbal or linguistic intelligence tests (but who frequently reach a higher level in the "performance tests" or "social maturity scale") who nevertheless possess sufficient capacity, not only to earn their living, but sometimes to achieve considerable success in some humble

²⁶ The conclusions of a very limited study may be briefly noted. Based on the Wells Alpha scores made by "768 white enlisted men selected on the basis of Army General Classification Test (AGCT) scores, as recorded on their qualification cards, to yield a distribution by Army Grades like that for all inductees entering during 1943," Tuddenham has ventured the opinion that the soldiers of World War II rated higher in tested intelligence than those in World War I. He ascribes the superiority to greater familiarity of the soldiers of the later war with group testing, to improvement in health and nutrition, and, especially, to about two more years of education. Tuddenham, Read D., "Soldier Intelligence in World Wars I and II," *The American Psychologist*, February, 1948, 54-56.

and unskilled but essential form of employment and who could scarcely be diagnosed as feeble-minded from the social and economic points of view. In spite of the cumulative evidence of the second decade, the *Dictionary of Psychology* as late as 1934 continued the fiction that morons have a top intelligence age of twelve years.

(d) *The percentage definition.* Let us consider a certain percentage of the general population to be feeble-minded—"the lowest 2 or 3 per cent of the total population in ability," according to the Presseys; the lowest 3 per cent, according to Pintner and Paterson; or the lowest one-half per cent, according to Miner. This suggestion possesses value for purely statistical purposes, provided a reasonable ratio is adopted, but little or no value for clinical diagnosis. The 3 per cent estimate would yield a total of about 4,000,000 feeble-minded persons in the United States, an exaggerated estimate five or six times as large as the older estimates before the advent of intelligence testing. The estimate for school children has varied from about 4 per cent to about 1 per cent. The writer's estimate (using the term in the socio-legal sense), based on the examination of many thousands of children in many school systems, would not exceed 1 per cent. Burt includes the lowest 1.5 per cent of "the school population of the same age" in the London schools (who have a mental ratio of 69.4 and less), but this estimate is admittedly based purely on the circumstance that the London special schools at the time of the estimate provided accommodations for 1.5 per cent of the pupils within the eligible ages.

It is possible and profitable to determine the percentage of pupils who are below a certain intelligence ratio (I. Q.) in any school system, or the proportion in need of special instructional aid, but the number who are feeble-minded cannot be accurately determined by such expeditious or offhand methods.

C. *The socio-occupational definition.* From the legal or social and economic point of view, a mentally defective per-

son is one who is decidedly socially inadequate and vocationally incompetent or dependent because of intellectual defectiveness. He is unable to meet the social demands, to conform to the conventions, customs, practices, laws, and moralities of the community. He is incapable of subsisting by his own unaided efforts or of independently directing or managing his social and economic obligations. He is not self-directing. The definition as thus drawn, however, is too inclusive, because there are many reasons why people become social parasites. A differential diagnosis is needed to exclude persons who are social parasites for reasons other than intellectual deficiency. Not all social and economic inadequates can be classed as mental defectives, as indicated by the following classifications:

(1) Some are criminally inclined, although of good or fair mental capacity. They may be impulsive, emotionally unstable, excitable, weak, indolent, hypersuggestible, viciously inclined, depraved, lacking in moral stamina or ethical ideals, and the like.

(2) Some are psychopathic or outspokenly psychotic. They may possess good mental capacity and adequate insight and comprehension of moral situations, but they are so emotionally, temperamentally, or volitionally unstable as to be incapable of inhibiting their impulses and of controlling their conduct. Or they may be incapable of exercising such control because they are mentally deranged.

(3) Some may be physically incapacitated—paralyzed, blind, deaf—and therefore unable to maintain themselves without external support. However, the programs of vocational rehabilitation have demonstrated that the large majority of physical defectives can be made financially self-supporting and socially adequate.

(4) Some may be immature. A five-year-old child cannot support himself although he may be mentally normal or precocious.

(5) Some may have demented to a state of dependency—

they are dements rather than aments—whereas others may have become mentally deranged or aberrant although intellectually bright: they have lost their capacities for social adjustment and economic adequacy.

None of these types of socio-industrial disability is caused by mental deficiency, and they must be excluded in the differential diagnosis. Only when the social inadequacy or occupational inefficiency is due to defect of intelligence from birth or from early life, should it be referred to as mental defectiveness.

In England the problems of mental defect and defectiveness have been subjected to prolonged investigations for over half a century by official committees or commissions, the reports and recommendations of which have led to the formulation of legal or statutory definitions that have profoundly influenced the thinking and practice of the English-speaking world. Among the reports of particular significance are those of the Departmental Committee, appointed in 1897 by the Lord President of Council (acting as chief of the Education Department), whose report led to the enactment of the Elementary Education (Defective and Epileptic Children) Act in 1899 (modified somewhat in 1921), which provided the first legal definition in England of "mentally defective children," for whom special-class grants were made on a permissible basis in 1899 and on a mandatory basis in 1924; the Royal Commission on the Care and Control of the Feeble-Minded, appointed in 1904, and rendering a voluminous report in eight "blue books" in 1908,²⁷ which led to the Mental Deficiency Act of 1913,²⁸ providing statutory definitions of the three grades of mental defectives—the idiots, the imbeciles, and the feeble-minded (and also moral imbeciles)—patterned after the earlier definitions framed by the Royal College of Physicians; and the Mental Deficiency Committee of 1924 whose

²⁷ Radnor, J., *Report of the Royal Commission on the Care and Control of the Feeble-Minded*. London: His Majesty's Stationery Office, 1908.

²⁸ Davey, H., *The Law Relating to the Mentally Defective* (2d edition). London: Stevens & Sons Ltd., 1914.

investigations were in full swing when the amended Mental Deficiency Act of 1927 was enacted.²⁹ The latter incorporates minor revisions in the 1913 definitions, particularly with respect to the extension of the life-age range within which a mental defect from early developmental arrest or injury can be technically classified as a case of amentia (mental defectiveness) rather than dementia.

The term of the broadest connotation and widest application defined in the 1927 revision for the first time is "mental defectiveness," which is defined as "a condition of arrested or incomplete development of mind existing before the age of 18 years, whether arising from inherent causes or induced by disease or injury." In the 1921 revision of the 1899 Education Act for defective school children between seven and sixteen the definition was as follows: mentally defective children are those who, "not being imbeciles, and not being merely dull or backward are . . . by reason of mental defect incapable of receiving proper benefit from the instruction in the ordinary Public Elementary Schools, but are not incapable by reason of that defect of receiving benefit from instruction in . . . Special Schools or Classes." It is obvious that, based on the statutes, public school special classes in England are limited to "feeble-minded" children as defined in the Mental Deficiency Act of 1927, although high grade imbeciles and borderline cases undoubtedly are sometimes assigned to these classes.

The definitions of the different grades of mental defectives in the British Mental Deficiency Acts, which continue to direct our thinking, are fundamentally social categories, based upon the degree of social proficiency and independence and occupational competency evidenced by persons classified as mental defectives.

In view of the fact that the British statutory definitions of the different grades of mental defectiveness have, since the

²⁹ Wood, Arthur H., *Report of the Mental Deficiency Committee*. London: His Majesty's Stationery Office, 1929, Parts I to IV (especially Part II).

beginning of the century, consistently emphasized social incompetency as the root element of the concept, it is amazing to find that the most general 1927 definition makes no reference to this basic criterion. The definition is, in point of fact, so general and vague as to be worthless as an attempt at scientific definition. There are many degrees and kinds of "arrested or incomplete development of mind before the age of 18 years," most of which do not constitute "mental defectiveness" as a legal category, and some of which represent processes of deterioration rather than "arrested or incomplete development." The socio-occupational criterion, however, continues to occupy a central position in the definitions of the sub-grades of mental defectiveness:

Idiots are "persons so deeply defective in mind from birth, or from an early age, as to be unable to guard themselves against common physical dangers." Accordingly, the criterion of idiocy is lack of the power of self-preservation. Idiots are so gravely defective that they cannot guard themselves against ordinary dangers, cannot care for themselves, and are wholly dependent economically. The qualification of "common" physical dangers was added in the 1913 Mental Deficiency Act, and marks an improvement upon the 1908 definition—even the best of us have a time dodging the automobiles of this generation.

In the 1927 Act the definition now in force reads as follows: Idiots are "persons in whose case there exists mental defectiveness of such a degree that they are unable to guard themselves against common physical dangers." The revision marks no essential change except for the omission of the qualification "from birth, or from an early age."

In the Vineland Industrial Classification (p. 191) high grade idiots have less than a three-year Binet age, and their I. Q.'s in the Terman classification (p. 19) are "below 20 or 25." The British legal definitions of the different grades are not couched in terms of intelligence age or I. Q.

According to the 1913 Act *imbeciles* are "persons in whose

case there exists from birth or from an early age mental defectiveness not amounting to idiocy, yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so." The 1937 definition is essentially the same except for the omission of the age qualification: "persons in whose case there exists mental defectiveness, which, though not amounting to idiocy, is yet so pronounced that they are incapable of managing themselves or their affairs, or, in the case of children, of being taught to do so."

Both of these definitions omit one of the essential elements of the traditional concept, namely, imbeciles, although capable of self-preservation (that is, guarding themselves against ordinary dangers, such as avoiding hot stoves or walking into rivers), are capable of only partial support even under favorable circumstances. The general run of imbeciles can be trained to take care of ordinary personal needs, to do simple routine tasks, and to contribute a little toward their own support, with a minimum of supervision, but they cannot entirely support themselves even under very favorable circumstances. They are given a Binet age from three to seven (the writer would prefer to draw the upper line at six) and I. Q. range of from 20 or 25 to 50.

The *feeble-minded* (high grade mental defectives, roughly corresponding to the American *morons*), according to the 1913 Act, are "persons in whose case there exists from birth or from an early age mental defectiveness not amounting to imbecility, yet so pronounced that they require care, supervision, and control for their own protection or for the protection of others, or, in the case of children, that they, by reason of such defectiveness, appear to be permanently incapable of receiving proper benefit from the instruction in ordinary schools."

In terms of Binet ages, they have been given a range of from eight to twelve years, and in terms of I. Q.'s a range of from

50 to 80. The correctness of the upper limits is questioned in a later section.

COMMENTS on this definition of feeble-mindedness (moronity). Millions of children of backward and borderline levels of intelligence exist who cannot "receive proper benefit" from the regular curriculum or in the ordinary grades but cannot, by any stretch of the imagination, be considered as feeble-minded, socially considered. In fact, the large majority of public school children who cannot receive "proper benefit from the instruction in ordinary schools," are backward or dull normal, rather than feeble-minded. The rest of the definition constitutes a marked improvement on the old form which has persisted in spite of its rejection by England in 1913. According to the 1908 definition, a feeble-minded person is "one who is capable of earning a living under favorable circumstances, but is incapable, from mental defect existing from birth, or from an early age (*a*) of competing on equal terms with his normal fellows; or (*b*) of managing himself and his affairs with ordinary prudence." This definition contains two fatal defects. First, a multitude of persons exist who cannot compete "on equal terms with their normal fellows," who are not feeble-minded but merely backward or dull. Second, the concept of "ordinary prudence" is unscientific and impracticably vague. Many persons exist who, in the judgment of others, cannot manage their affairs with ordinary prudence, who certainly are not sufficiently deficient to be classed as feeble-minded, and, in fact, may be quite intelligent. This definition emasculates the concept of feeble-mindedness for it would be possible to class almost all kinds of social ne'er-do-wells as feeble-minded—as has, indeed, often been done. The revised definitions emphasize the point of social significance, namely, that the "mental defectiveness" is "so pronounced that they require care, supervision, and control for their own protection or for the protection of others." To quote one of Tredgold's earlier statements: the term feeble-

mindfulness "should be restricted to those persons who are so lacking in general mental capacity, in common sense, that they are incapable of subsisting by their own unaided efforts. . . . There are many individuals who, although scholastically dunces, have yet sufficient aptitudes of other kinds and, in particular, sufficient common sense, not only to take care of their interests, but to achieve a considerable degree of success in a humble walk of life."³⁰ Doll also says, "Mental deficiency . . . is typically defined as a condition such that the person affected is (a) socially insufficient, because of (b) subnormal intelligence, (c) existing from an early age. Whatever else might be added to the description these three elements are essential."³¹

The difficulty of making a prognosis of social inadequacy or incompetency in the socio-legal sense in the case of children should be clearly recognized. Granting that the level of intelligence has been established with reasonable accuracy, the ability to make satisfactory social and economic adjustments depends on many other factors than intelligence—nervous stability, emotional poise, industry, determination, willingness to work, trained skill, the keenness of the competitive labor market, availability of suitable types of employment, and the complexity of the social and industrial environment. Perhaps it depends to no small extent on the subject's capacity to deal with things and persons, his social age and mechanical ability. It is Doll's contention that the ability measured by the Vineland Social Maturity Scale correlates more closely with social adequacy and occupational success than the traits assessed by the Binet scale. "Social quotients by this scale which fall below 70 (uncomplicated by specific handicaps) suggest strong suspicion of mental deficiency, while those

³⁰ Tredgold, Alfred F., *Mental Deficiency*. Baltimore: William Wood & Company, 1920, p. 385.

³¹ Doll, Edgar A., "Notes on the Concept of Mental Deficiency," *American Journal of Psychology*, January, 1911, 116-124.

above 80 give a presumption of normality."³² The upper limit of idiocy is placed at a social age of three years, imbecility at nine, and morosity at eighteen. This upward extension of the thresholds of imbecility and morosity constitutes a challenge for further research.

According to Doll, mental defectives reach intellectual maturity earlier than social maturity.³³ Thus idiots reach intellectual maturity at six or eight, imbeciles at ten or twelve, and morons usually by fifteen. On the other hand, social maturity is reached by the imbeciles at about fifteen, and by the morons at about twenty.

ESTIMATES. The ratio of mentally defective persons (from the socio-legal standpoint) in the general population and the ratio of the different grades in exact numbers classifiable as mental defectives from the standpoint of socio-occupational competency is unknown, partly because of the difficulty of definition, partly because of the difficulty of determining whether young retardates will ultimately prove to be feeble-minded, and partly because of the difficulty of examining all of the inhabitants of a given state or major region. Only estimates based on surveys in limited areas are available. The most extensive investigations are those made by the British Commissions. The estimate of the 1904 Royal Commission was 4.6 per 1,000 of the general population for England and Wales, of whom 5 per cent were idiots, 20 per cent imbeciles, and 75 per cent feeble-minded (morons).³⁴ Twenty years later Edmund O. Lewis,³⁵ reporting for the Mental Deficiency Committee, found at least 8 mental defectives per

³² Doll, "Notes on the Concept of Mental Deficiency," 120. See also "The Nature of Mental Deficiency," *Psychological Review*, September, 1940, 395-415. In this article the author makes a case for the view that the difference between the normal and the mentally defective is qualitative, so far as concerns the special clinical types.

³³ Doll, "The Feeble-Minded Child," in *Manual of Child Psychology*, Carmichael, Leonard. New York: John Wiley & Sons, Inc., 1946, pp. 845-885.

³⁴ Tredgold, *Mental Deficiency*, pp. 12-22.

³⁵ *Report of the Mental Deficiency Committee*, Part IV, pp. 54 ff.

1,000 for England and Wales (about one-half children and one-half adults), of whom 25 per cent were imbeciles and idiots and 75 per cent were feeble-minded. Both commissions found more mental defectives in the rural than in urban areas, the incidence in the Lewis report being 6.7 per 1,000 in the urban areas and 10.49 in the rural areas. Both commissions reported a preponderance of males, the ratios in the Lewis report being 9.21 males to 7.97 females per 1,000 of each sex. Estimates reported from the United States between 1914 and 1917 varied from 3.40 (a New York investigation) to 7.35 (Porter County, Indiana Survey) per 1,000. In World War I, 12 per 1,000 were rejected from the American Army because of mental deficiency.³⁶ Of 7,907 first admissions to public institutions for mental defectives in 1943, 14.2 per cent were classified as idiots, 29.2 per cent as imbeciles, and 44.9 per cent as morons; 11.6 per cent were unclassified.

D. *Educational definition.* Broadly stated, mental defectiveness may be characterized educationally as the inability, after years of instruction or protracted drill, to acquire any considerable or useful mastery in the literary subject matter, especially written composition, reading, and arithmetic. What is the highest school grade that the ablest mental defectives can reach? Based on investigations of the educational accomplishments of various grades and types of subnormals in the St. Louis Special Schools, very few of whom were over fourteen years of age, the conclusion was reached that genuinely mentally defective children (diagnosed conservatively on the basis of multiple criteria) cannot do successful all-around work in the literary branches beyond the latter part of the second grade or the first part of the third grade.³⁷ In

³⁶ Ireland, M. W., *Defects Found in Drafted Men*. Washington: Government Printing Office, 1920; *Neuropsychiatry*. Washington: Government Printing Office (The Medical Department of the United States Army), 1929, Vol. X.

³⁷ Wallin, "The Pedagogical Status of the Feeble-Minded Children," *The Elementary School Journal*, January, 1918, 588-597; "The Achievement of Mental Defectives in Standardized Educational Tests," *School and Society*, 1919, 250-256; "The Achievement of Subnormal Children in Standardized Educational Tests," *Miami University Bulletin*, 1922, XX, No. 7.

a survey of 600 London special school children, Burt ³⁸ "found only 7 per cent up to and none above the level of standard II. This is equivalent to a scholastic age of eight." Walter B. Cornell, after examining mental defectives in the Philadelphia schools, concluded that "the high-grade feeble-minded as a rule stick fast" in "the third school grade." In the special schools of Birmingham, England, the ablest pupils at the age of sixteen were "able to read and calculate to about the same extent as a normal child of eight or nine," according to Mrs. Hume Pinsent. Tredgold concluded that none of the mental defectives of twelve years in a London special school reached normal standard II scholastic requirements. "In occupational and manual work they are decidedly better." ³⁹ During the second decade the statement was often reiterated in this country that the high grade morons stuck fast in the fifth or sixth grades. It is probable that most of these children had been automatically promoted beyond their level or were dull normals. Of course, some mental defectives possess special abilities and may reach higher levels in one or more branches. Most mental defectives, fortunately, possess considerably higher ability in manual and occupational work.

RETARDATION CRITERION. How much educational retardation is indicative of feeble-mindedness? Miner suggests as a "low" estimate five or more years of retardation for children twelve years of age or older, which would place them in the first or second grade, and as a "maximum" estimate four or more years of retardation for the children of the same ages, which would be equivalent to second- or third-grade capacity.

RATE OF PROGRESS CRITERION. What rate of progress do the mentally defective make in school? Shrubsall estimates that the "true defective" advances at less than half and the "merely backward" at more than half of the normal rate. Of course, the rate of improvement varies enormously with the degree of deficiency. With possibly one or two exceptions, we have

³⁸ Burt, *Mental and Scholastic Tests*, p. 168.

³⁹ Tredgold, *Mental Deficiency*, 1914, p. 169.

never known imbeciles who could do satisfactory all-around first-grade work after six or seven years of schooling. The ablest imbeciles made less than 15 per cent of progress. On the other hand, the highest grade of the mental defectives progressed only about one-third as rapidly as the normal children. They required about three years to do one year of work in the literary curriculum.

VALUE OF THE DEFINITION. The educational criteria suggested for the diagnosis of mental defectiveness are far from infallible, nor are they even reasonably reliable, owing to the fact that a child's rate of progress and possible attainments in school depend on many factors other than inherent ability. Nevertheless, the educational symptomatology should never be disregarded in the study of mental deficient. Lack of successful progress in school is the most frequent reason for referral of children to the psychoeducational clinic, and mental defectiveness is one of the most frequent causes of academic failure.

E. *Eugenic definition.* Feeble-mindedness sometimes represents a condition of racial or biological inaptitude or eugenic unfitness. Some of the mentally defective are produced by a cacogenic heredity, and some of them breed true; some transmit their strains to posterity; some cannot procreate a normal, healthy progeny. The dysgenic efforts are particularly evident when two mentally defective persons of the familial type mate. Although the marriage of all feeble-minded persons may not prove dysgenic—normal persons have been born of mentally defective parents—marriage of the mentally defective should be interdicted, because they make inefficient, incompetent, and more or less irresponsible parents and breadwinners.

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Chapter 3

PSYCHOLOGICAL CONCEPTS AND THEORIES OF MENTAL DEFECTIVENESS

The two chief points in question in the theories of mental defectiveness are: First, do mentally defective persons differ psychologically from normal persons quantitatively or qualitatively? Second, is mental defectiveness a simple or a complex state or condition? If it is a simple condition, the problem of diagnosis should be relatively simple and it should be possible to formulate a working definition in simple, unambiguous terms, and a simple, comprehensive classification. On the other hand, if the condition is very intricate and complex, the problems of diagnosis, definition, and classification become complicated and difficult of formulation in unequivocal terms. What are, briefly, the essential postulates and implications of the rival hypotheses?

THE QUALITATIVE VERSUS THE QUANTITATIVE THEORY

The qualitative theory

Binet and Simon, the outstanding proponents of the qualitative theory, held that the "defective" child differs from the normal child in the quality or kind of his mental make-up.¹ "A defective child does not resemble in any way a normal one whose development has been retarded or arrested. He is inferior, not in degree, but in kind." If this view is carried

¹ Binet, Alfred, and Simon, T., *Mentally Defective Children* (Translated by W. B. Drummond). New York: Longmans, Green & Company, Inc., 1914, p. 13.

to its logical extreme, the mentally defective or deficient child must be regarded as a different species or type of being from a normal one.

Argument based on scattering. One of Binet's chief arguments was that the retardation in the mentally defective is not uniform or equal in all directions. "So far as certain faculties are concerned, he remains on the level of a younger child; but in respect to others, he is on the level with normal children of his own age."² This "want of equilibrium" is the "specific characteristic" or the "differentiating attribute of the defective child."

In harmony with this doctrine attempts have been made to diagnose "potentially feeble-minded" children (and psychopathic cases) on the basis of extensive scattering in the Binet-Simon scale. A child who scatters widely on the scale passes some tests over a wide range of years above the basal age, possibly in a half-dozen age levels, while he may also fail on some tests below the basal age. Wide scatter indicates intellectual irregularity or disharmony of intellectual functions, or lack of equilibrium in the reactions to the tests in the scale. A "potentially feeble-minded" child is one who does not test as sufficiently backward at the time of the examination to justify a diagnosis of feeble-mindedness, but who is so seriously lacking in the potentialities for normal development that he eventually stagnates as feeble-minded. To quote Doll, "In the Binet tests the typical normal child has a basal year not more than one year below his chronological age and passes few tests beyond his chronological age. The potential feeble-minded, on the other hand, has the basal year more than one year below the chronological age," and "scatters, failing in tests one would expect a normal child of that age to pass, and succeeding in others not expected."³

² *Ibid.*

³ Doll, "Preliminary Note on the Diagnosis of Potential Feeble-Mindedness," *Training School Bulletin*, May, 1916, 54-61.

Scattering not excessive among mental defectives. The crucial test of this hypothesis is, do mentally defective children, in point of fact, vary more in different mental traits than do normal or backward children? Binet and Simon, unfortunately, did not produce conclusive proof that they did. They merely described a few imbeciles who evidenced great irregularity in their mental make-up. The wide generalization they reached went far beyond their premises. It was not based on a detailed analysis of a wide array of psychological tests administered to a large number of children of various chronological ages and levels of intelligence. On the basis of a very simple measure of scatter (the number of tests or months passed beyond the basal age) in the 1908, 1911, and Stanford Binet-Simon 1916 scales among 2,206 examinees of various kinds and ages (normal, backward, borderline, moronic, imbecilic, epileptic, insane, neurotic, psychopathic, unstable, and delinquent), mostly juveniles, the conclusion was reached that there is no warrant for the assumption that "unequal development, lack of uniformity, or scattering in intelligence is the specific characteristic, the differentiating attribute, the pathognomonic sign of feeble-mindedness or potential feeble-mindedness."⁴ This conclusion was later confirmed by Pressey and Cole, Mathews, Brown, and Gilliland, Wittman, and Goldman. The greater scattering among the mentally retarded found by Doll, Merrill, Fox, Woodrow, and Shipley is not sufficiently marked to be statistically significant or of any aid in the diagnosis of feeble-mindedness.⁵ The

⁴ For the factual data consult the following study and the references to the earlier investigations contained therein: Wallin, "A Further Note on Scattering in the Binet Scale," *Journal of Applied Psychology*, April, 1927, 143-154.

⁵ For summaries of the literature on scatter see Harris, Albert J., and Shakow, David, "The Clinical Significance of Numerical Measures of Scatter on the Stanford-Binet," *The Psychological Bulletin*, March, 1937, 134-150; and "Scatter on the Stanford-Binet in Schizophrenic, Normal, and Delinquent Adults," *Journal of Abnormal and Social Psychology*, January, 1938, 100-111; Mayman, Martin, "An Analysis of Scatter in Intelligence Test Results: A Review of the Literature," *Transactions of the Kansas Academy of Science*, March, 1946, 48: 429-444.

data on the extent of scatter among other kinds of abnormal individuals, such as epileptics, psychopaths, delinquents, and different types of psychotics, are somewhat discrepant, depending largely perhaps on the particular tests used.⁶

Emotional imbalance. Of course, Binet might have retorted that the lack of equilibrium to which he referred was primarily emotional or temperamental rather than intellectual. Obviously, such a conclusion would have been based on subjective impressions as no accurate measuring scales of emotional irregularities or disequilibrium existed in Binet's time. His criterion for want of equilibrium was, in all probability, intellectual and not affective or emotional, since his scale for identifying "defective" children was intellectual rather than emotional.

When, however, the invalidity of the twelve-year upper threshold of feeble-mindedness or mental deficiency and of the concept of high grade morosity became apparent to many psychologists from the results of the army psychological testing in the first world war, the theory was advanced that a person could be emotionally feeble-minded although intellectually normal or near normal.⁷ Under this expansion of the concept, individuals could be considered feeble-minded who are emotionally and temperamentally unstable and who suffer from defective powers of emotional and volitional control—defective inhibition—although they may be merely backward in intelligence, or even intellectually normal or supernormal.

CRITICISM. The following criticism of this extension of the concept was voiced a year after it was advanced:

⁶ *E.g.*, Gilliland, Wittman, and Goldman's results with the Wechsler-Bellevue scale of intelligence are largely negative: "Patterns and Scatter of Mental Abilities in Various Psychoses," *Journal of General Psychology*, October, 1943, 251-260. Roy Shafer reports greater scatter or disharmony of mental functions among schizophrenics than among normals on the Babcock Efficiency Tests: "The Significance of Scatter in Research and Practice of Clinical Psychology," *Journal of Psychology*, July, 1944, 119-124.

⁷ Miner, James B., *Deficiency and Delinquency*. Baltimore: Warwick & York, Inc., 1918. *Report of the Mental Deficiency Committee*, IX, pp. 112, 115, 139. Burt, Cyril L., *The Subnormal Mind*. London: Oxford University Press, 1935, p. 71.

First, it flies in the face of the historically fixed connotation of the term. Binet and Simon emphasized in unmistakable language that no one can be considered feeble-minded no matter how little he knows unless he is defective in judgment, or in intelligence, which is the "fundamental organ" of the mind. "Idiocy," as Esquirol was the first to recognize, consists in the weakness of the intelligence. "The child is judged to be an idiot because he is affected in his intellectual development. This is so strikingly true that if we suppose a case presented to us where speech, locomotion, prehension, were all nil, but which gave evidence of an intact intelligence, no one would consider that patient an idiot."⁸ Therefore, in order to measure the degree of feeble-mindedness, it was necessary to measure only the level of intelligence and not the emotional or instinctive deviations. This conclusion led to the development of the Binet-Simon measuring scale of intelligence. Binet and Simon provided for another class of children who were morally unstable. They were referred to as unstable, or illbalanced, or undisciplined. Some of these are "mentally defective," but the majority have a "degree of intelligence superior to that of the defective." Moreover, while their intelligence "is in general below the average," about a third may not be "at all backward."⁹

Second, it is difficult, in fact quite impossible, to measure by uniform objective tests an individual's degree of "emotional," or "conative," or "temperamental . . . instability or deficiency." Our judgment as to an individual's emotional and temperamental peculiarities will have to depend largely upon extra-laboratory observations of his reactions in the home, school, street, playground, or shop. How deficient or abnormal must the individual be emotionally, volitionally, or temperamentally (or emotionally undeveloped, immature, or infantile) in order that we may consider him feeble-minded? Certainly no one can answer this question. Since we cannot use definite tests to measure emoto-conative feeble-mindedness, the answer will depend upon the subjective opinion of the examiner, based either on his own or on reported observations. That most widely variant deductions will be drawn from such observations is well known to every investigator.¹⁰

⁸ *L'Année Psychologique*, 1905, 163 f.

⁹ Binet and Simon, *Mentally Defective Children*, pp. 6, 8, 15 ff.

¹⁰ Wallin, "The Concept of the Feeble-Minded, Especially the Moron," *Training School Bulletin*, May, 1920, 50 f.

Since this criticism was lodged, a number of tests or scales have been devised for the measurement of emotional maturity or instability or emotional or neurotic abnormalities of various kinds, among them projective techniques, such as the Rorschach and the thematic apperception tests, which have been used to a slight extent for the diagnosis or more accurate characterization of mental defectiveness.

Emotionally produced intellectual limitations and the diagnosis of mental defectiveness by projective techniques. It has been recognized for long that some children of normal intellectual endowment may test mentally defective by psychometric tests, such as the Binet or Wechsler-Bellevue, because of emotional disturbances or blockages (pseudomental defectiveness). It is maintained by some projective-technique examiners that such cases can be reliably distinguished from genuine mental defectiveness by means of "projective" examination methods (the term projective was suggested by Lawrence K. Frank), that many found defective by psychometric tests possess normal intellectual potentials as evidenced by projective techniques (especially the Rorschach), and, in fact, that the projective tests are superior to the psychometric for the diagnosis of mental deficiency.

EXPRESSIVE AND PROJECTIVE TECHNIQUES. These techniques include certain procedures in which the examinee is permitted and encouraged to respond as he sees fit and without any restraint to the stimulus situations presented by the examiner. The laboratory situation is so arranged as to call forth the subject's free, spontaneous responses on the assumption that the spontaneous responses will reveal or express his inner drives, impulses, motivations, attitudes, underlying cravings or wishes, fears, hostilities, conflicts, complexes, and the dynamics of his personality organization. It is also thought that his uninhibited responses may supply the clue to the interpretation of his earlier experiences. The projective technique, in the more limited connotation, refers to the pro-

jection of the subject's personal problems and characteristics into the characters of his play activities or into the interpretation of the stimulus situations. His fantasies are the expression of his personality problems and motivations. Some of the techniques are also used for therapeutic purposes, particularly for relieving the subject of his conflicts, tensions, fears, anxieties, and hostilities.

Many expressive techniques have been in use, some for a long time, such as responses to a list of discrete words, usually presented orally (Carl G. Jung's association-reaction method), the free-running or continuous association test (starting with a given key-word), the measurement of respiration and vasomotor changes, automatic writing, interpretation of pictures, sentence and story completion tests, sketching, drawing, brush painting and finger painting, modeling, the use of play materials (dolls), puppet shows, the dramatic representation of problem situations, dramatic plays, the psychodrama, and the perceptual and imaginal reaction to unstructured or ambiguous visual or auditory stimuli.

The most frequently used projective technique makes use of ten unstructured (meaningless or ambiguous) ink blots on cardboard devised by the Swiss psychiatrist, Herman Rorschach, in 1921. Some of the blots are in white and black, with variations in gray; some are in colors of varying saturation. The blots are exposed in definite order and the subject is asked to state orally what he sees in each blot as the card is shown or "anything that might be represented there." The investigator records the spontaneous responses and also the answers to the questions that are asked after all the cards have been shown. The examinee's peculiar "test profile" is supposed to reveal his inner tendencies, personality structure, idiosyncrasies, abnormalities, and dynamic mechanisms. The scoring, based upon the pattern of the responses, is usually in terms of certain formal categories, about 28 in all, such as whole or part responses, movement responses, and popular

responses. Beck has distinguished four characteristic patterns: that of the healthy adult of superior ability, the schizophrenic, the brain-injured, and the feeble-minded.¹¹

This is not the time or the place in which to attempt an evaluation of the Rorschach technique, which is attracting as much attention today as the Binet test did a third of a century ago and is in need of the same critical evaluation to counteract the exaggerations of its enthusiasts as the Binet required in an earlier generation. Rorschachists insist that no one is competent to administer the test and interpret the responses without extensive training, and such training is being provided in short courses in "Rorschach Institutes." Great need exists for ridding Rorschachism of its pedantic jargon and its atmosphere of cultism coupled with the implication that the successful practitioner must possess almost esoteric powers of interpretation.¹² After all, the Rorschach is only one among many psychological tests and must conform to the same process of scientific exploration and verification as any other method. The value that the test possesses will remain after it has been stripped of its occultism. In general, the difficulty and ambiguity of the interpretation of the responses, conceded by some Rorschachists, is much greater than with psychometric techniques.

A few recent applications of the Rorschach to alleged mental deficientes which yielded somewhat discrepant results may be cited. In a limited Rorschach study of cases committed to an institution for the mentally defective, 24 with high Wechsler-Bellevue I. Q.'s (median 85) and 26 with low I. Q.'s (median 67), Sloan found that the "five most frequently deviating factors" seemed to indicate the "absence of mental deficiency," and that the "deviations from the pattern of mental deficiency were found to be comparable, quantitatively, for both groups." But no Rorschach signs were proposed that

¹¹ Beck, Samuel J., "The Rorschach Experiment: Progress and Problems," *American Journal of Orthopsychiatry*, 1945, 15: 520-524.

¹² Thurstone, Louis L., "The Rorschach in Psychological Science," *Journal of Abnormal and Social Psychology*, October, 1948, 471-475.

are indubitably indicative of mental defectiveness.¹³ In a subsequent study of 15 pairs of mental defectives on extra-institutional wage placement, Sloan found that quantitative statistical comparisons of Rorschach factors "failed to distinguish successful from unsuccessful wage placement." The test did not prove valuable for the prediction of extramural adjustment.¹⁴

Jolles administered the Rorschach to 34 children above ten years of age whose I. Q.'s were below 80 as determined by the 1937 Stanford-Binet (with the Wechsler performance or the Arthur or Cornell-Coxe performance scales) or the complete Wechsler. The children had been referred from various school districts in Illinois to the State Department of Public Instruction for certification for special classes for the mentally retarded.¹⁵ All the children were classified as cases of "familial or undifferentiated mental deficiency," without any "known neurological defects resulting in mental deficiency." A few illustrative case histories were supplied, but no tabular data. Jolles maintains that every one of the examinees was a case of "severe emotional maladjustment of some type," a conclusion apparently based primarily upon the Rorschach blots. In fact, on the basis of the Rorschach, 21 children were classified as cases of "anxiety neurosis" and 8 as "schizoid personalities"; that is, 29 out of 34 cases were essentially psychopathic cases rather than mental defectives. The general conclusion of the investigation was that "mental deficiency of the familial and undifferentiated type (which includes the majority of mental defectives) is a symptom of personality disorder rather than an indication of limited mental ability," an enormous "inductive leap," based primarily upon verbal reactions to a few ink blots.

¹³ Sloan, William, "Mental Deficiency as a Symptom of Personality Disturbance," *American Journal of Mental Deficiency*, July, 1947, 31-36.

¹⁴ Sloan, William, "Prediction of Adjustment of Mental Defectives by the Rorschach," *Journal of Consulting Psychology*, September-October, 1948, 303-309.

¹⁵ Jolles, Isaac, "A Study of Mental Deficiency by the Rorschach Technique," *American Journal of Mental Deficiency*, July, 1947, 37-42.

In a later and more complete study, which contains additional case records for the same group of pupils, this general conclusion is reaffirmed and the prediction is made that "many mental defectives may be treated successfully by psychotherapeutic techniques."¹⁶

In the attempt to evaluate the findings and claims of this investigation a few relevant facts should be kept clearly in mind.

(1) It does not appear from the record that the investigator had any special training in the administration of the Rorschach or in the interpretation of Rorschach results, the importance of which has been repeatedly emphasized by the Rorschach practitioners. He had a general acquaintance with Samuel J. Beck's publications.

(2) Many of the examinees referred to as "mental defectives" in the title and elsewhere in the articles were not mental defectives in the technical sense of that word, according to the data supplied from the psychometric tests. Many of them were merely backward or dull normal.

(3) The existence of large differences or discrepancies between the Rorschach and the psychometric tests—and many such differences exist—does not demonstrate that the findings from the psychometric tests were all spurious and that those from the Rorschach results were more accurate and reliable. Similar preposterous claims regarding the infallibility of the Binet tests were made by some of the devotees of the Binet in 1910. The role of the examiner's subjectivism in the interpretation of the examinee's responses is greater in the Rorschach than in the Binet or similar psychometric tests. The same extreme claims are now being made for the Rorschach as were made for the Binet in the flush of its success.

¹⁶ Jolles, "The Diagnostic Implications of Rorschach's Test in Case Studies of Mental Defectives," *Genetic Psychology Monographs*, November, 1947, 89-197. The writer states that his study is unique because the "mental defectives that have been examined are noninstitutional children." In point of fact, all of the numerous investigations conducted by the author have been based upon noninstitutional cases except groups of epileptics and psychotics examined between 1910 and 1912.

The danger is in assuming that the Rorschach possesses magical powers and in placing too great a diagnostic burden upon it. The Rorschach is no more of a magical or infallible diagnosticon than is the Binet or any other psychometric test. In the measurement of ability errors occur with the Rorschach as frequently and as extensively as with the Binet and any other similar test, in fact, the errors probably are even greater.

(4) Jolles does not present any evidence that his functional or emotional "mental defectives" were or could be restored to intellectual normality by "psychotherapeutic techniques." The work of Despert and Pierce,¹⁷ to which he refers, has been questioned by Thompson and Harris.¹⁸ Emotional therapy may be of value for mental defectives as for normal persons, but is it a cure-all for mental defectiveness?

(5) As a check, the author would suggest that all of Jolles's putative cases of "anxiety neuroses" and "schizoid personalities" be carefully examined by a thoroughly competent neuropsychiatrist. Has anyone shown that the possession of anxiety or schizoid states is a guarantee of immunity against mental defectiveness? Does not the evidence show that mental defectives, no less than normal persons, may become victims of psychoneuroses and psychoses? That many mental defectives manifest emotional and neuropathic disorders is freely admitted; but that all mental defectiveness of the primary or endogenous type can be reduced to emotional disturbances or inhibitions imposes too great a strain upon one's credulity. The Rorschach may illuminate some of the dark recesses in the subnormal mind; it may add a scintillating facet to the portrait; but that it supplies a satisfactory substitute for psychometric procedures for determining grades of

¹⁷ Despert, J. L., and Pierce, Helen O., "The Relation of Emotional Adjustment to Intellectual Function," *Genetic Psychology Monographs*, 1946, 34: 3-56.

¹⁸ Thompson, Clare W., and Harris, Robert E., "The Relation of Emotional Adjustment to Intellectual Function—a Note," *Psychological Bulletin*, 1947, 283-287.

mental deficiency will remain an assumption until the proofs are forthcoming.

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For the application of another projective test to mental deficient see Sarason, Seymour B., "The Use of the Thematic Apperception Test with Mentally Deficient Children," *American Journal of Mental Deficiency*, October, 1945, 272-276.

The quantitative theory

According to the second theory, advocated by Edward Seguin, Francis Galton, Edward L. Thorndike, and many other psychologists, the fundamental psychological difference between the normal and the mentally defective is one of degree, amount, or quantity, rather than one of kind. The mentally defective and normal belong to the same genus *homo*; they have the same basic mental traits or capacities, but the mental defectives are less richly endowed. They possess less judgment, less general intelligence, less understanding, less initiative, less adaptability, less power of attention than the normals. The mental functions that they do possess are essentially the same as those possessed by the rest of us, but they have them in smaller amounts.

Many investigations have been concerned with the determination of the differences that exist in the mental traits of mentally defective, dull, normal, and bright children, and many qualitative differences have been postulated. Table II summarizes certain differences between bright and dull children, as revealed in the Baltimore study.¹⁹

¹⁹ *Five Unifying Factors in American Education*, Part I, "Pupil Promotion Problems." Washington: Department of Superintendence of the N. E. A., Ninth Yearbook, 1931, p. 108.

Concededly, the sharp dichotomies apparent in the contrasting traits in this tabulation appear to be genuine qualitative differences. But the dichotomies are largely spurious, for the superficial qualitative differences can ultimately, so it seems to us, be resolved into quantitative differences. To illustrate: when we say that the feeble-minded child possesses

TABLE II

Characteristic Differences of Dull and Bright Pupils
as Recorded in a Baltimore Investigation

<i>Dull</i>	<i>Bright</i>
Inability to coordinate two or more mental functions	Ability to coordinate any number of functions
Difficulty in assimilation	Ease of assimilation
Learning through detailed, simple material	Ability to generalize, pick up clues from less concrete materials
Short attention span	Sustained attention
Slow reaction time	Quick reaction time
Restricted curiosity and limited initiative	Intellectual curiosity and initiative
Limited imagination	High imagination
Personal viewpoint	Broadminded, impersonal attitude toward problems
Dependence on criticism and approval of others	Self-criticism; intellectual approval highly satisfying
Lack of appreciation of intellectual humor	Sense of humor
Trouble with language symbols	Keen language ability
Narrow interests	Versatility and vitality of interests
Slow reading habits	Rapid reading habits
Dependence on others to show application of previously learned experiences	Sensitivity to application of knowledge
Inadequate memory for abstractions	Logical and accurate memory
Observation without generalization	Close observation
Immediate recall	Delayed recall
Judgment on inadequate data	Ability to reason without going through concrete experiences; suspended judgment until necessary data are in
Better performance through repetition without much attention to principles	Better performance through understanding principles
Emotional bias in action	Rationalized feelings
Insensitivity to intellectual and esthetic elements in a situation, unless pointed out by others	Sensitivity to intellectual and esthetic elements in a situation

All persons are psychologically classifiable linearly. The first corollary of the quantitative theory is that all human beings can be arranged psychologically, that is, from the psychometric standpoint, in a straight line from poorest to best, just as all human individuals can be similarly arranged with respect to any physical trait. Thus, if they are arranged in the order of height, all adults will occupy relative positions between a minimum of fifteen inches (the measurement of the shortest reported adult, a female dwarf, which is less than the length of the average child at birth), and a maximum of nine feet and four inches (the Finnish giant referred to on p. 321). The line connecting these extremes would probably be a straight oblique if a sufficient number were measured.

Psychologically, human individuals can be similarly arranged in a straight line, either with respect to a single or relatively simple trait, such as the speed of reaction, adding columns of digits, or crossing out A's in columns of letters, or the accuracy of trap shooting; or a combination of traits or a complex trait, such as general intelligence.

PSYCHOMETRIC CLASSIFICATION OF INDIVIDUALS. So far as concerns general intelligence (or general mental capacity or alertness, as it is sometimes called) mankind can, according to the quantitative theory, be arranged along one axis from the least gifted, the profound idiots, at the lower extreme to the ablest, the most brilliant, at the upper extreme. All other humans are ranged between these extremes.

Fig. 1 contains a simple, practical scheme of distribution of general levels of capacity based on the psychometric concept of grades of ability. The linear surface of distribution is divided into three main classifications, as indicated by the divisions above the horizontal line, with the minus deviations on the left and the plus deviations on the right of the average or typical measure, and into ten or more minor subdivisions, as shown by the designations below the horizontal.

The terms subnormal, subaverage, or inferior, and super-normal, superaverage, or superior are used generically, the

former embracing all degrees of deficiency below normal, and the latter all degrees above normality. The term normal is not used in the sense of a sharp mathematical point on the curve of distribution, but as a broad range which includes persons somewhat below or somewhat above the mathematical average. A generous public is not inclined to be very exacting in the application of the concept of normality to minor deviates or even to deviates that impress the expert as clearly abnormal.

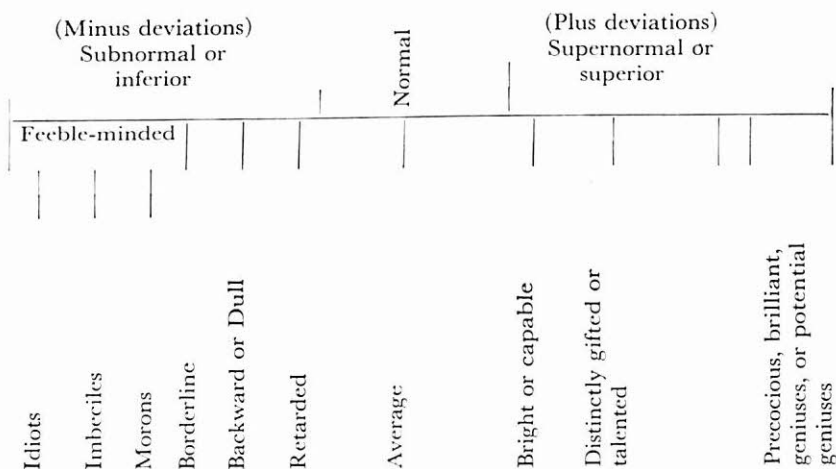


FIG. 1. Surface of distribution of human beings according to general ability.

The terms below the horizontal indicate ever increasing degrees of deviation from the central tendency. All on the minus side of the curve are subnormal in various degrees and all on the plus side are supernormal in various degrees. But the persons at the extreme ends, the idiots and the geniuses, belong to the same human species. In fact, the curves of intelligence capacity of the infrahumans and humans overlap. Many quadrupeds and infrahuman primates (apes and gorillas) possess more ability and are more amenable to training than many idiots and imbeciles. To constitute a distinct species, the probable error (P. E.) in psychological test scores for idiots would have to exceed 5 "from the median for or-

dinary people.”²⁰ Norsworthy found a P. E. of -7 in the intelligence tests, -3.5 in the memory tests, and -2.7 in the maturity records. In every test some defectives equaled the median for ordinary children.

Different authorities draw the boundary lines between the different minor subdivisions at different points in the scale of intellectual ability and do not apply the same terminology to the different groups. For example: the word retarded is here arbitrarily used to denote a slight degree of intellectual shortage scarcely distinguishable from intellectual normality which can frequently be removed by proper orthogenic procedures, whereas others have employed the term as a euphemistic equivalent of mentally deficient. Our use of the term backward doubtless corresponds to the term dull normal, as used by some writers. Our knowledge of those on the subnormal side of the curve is probably even now more complete than of those on the supernormal side, partly because the subnormals have been intensively studied for a much longer period of time (at least since the days of Jean Marc Gaspard Itard, French physician to an institution for the deaf in Paris, who in 1798 began his four-year period of scientific investigation and training of the “wolf boy” referred to on page 134), and partly because it is easier to characterize and classify inferior minds than brilliant ones. The supernormal or superior mind is far more complex and elusive than the subnormal one. Its greater subtlety of operation will probably defy minute analysis by the clumsy methods of investigation now available. The mind of the genius will probably remain more or less inscrutable and unclassifiable even after our diagnostic techniques have been improved. The growth and functioning of the subnormal mind, on the other hand, affords unusual opportunities for analytic study because of its relative structural simplicity and its slowness of development. The opportunity for study which it affords can be likened to the improved

²⁰ Norsworthy, Naomi, *The Psychology of Mentally Deficient Children*. New York: Science Press, 1906, pp. 77-85.

observation of pictures possible with slow motion photography. However, most authorities are agreed that mankind can be arranged in rank order with respect to general mental capacity in conformity with the necessary requirement of the quantitative theory.

The curve of distribution of intelligence is an unbroken continuum. The second consequence of the theory is that no sharp or distinct lines of demarcation can be drawn between the different groups or subdivisions of the intelligence classification. That is, there are no gaps or sharp divisions between the normal and retarded, between the retarded and backward, between the backward and borderline, between the borderline and high grade mentally defective, and so on. The different groups shade insensibly into one another. They are merely different grades or degrees of the attribute in question and not distinct types. On the qualitative theory we should expect to find a distinct gap or line of cleavage, at least between the mentally defective and the not mentally defective. But the conceded impossibility of making infallible diagnoses of the high grade mentally defective, or even measurably satisfactory differentiations between the high grade mentally defective and the borderline, proves that no such line of demarcation exists in nature. The task of diagnosing mental defectiveness would indeed be simple if a definite break existed between the mentally defective and those not mentally defective, and diagnostic disagreements between different examiners would not exist. However, the existence of such disagreements is notorious. The existence of overlapping and insensible gradations accounts for the diagnostic confusions or divergencies in the ability groupings or classifications of different diagnosticians. Man differentiates and classifies from different points of view and arranges facts into separate and distinct groups and categories in accordance with predetermined purposes, in order to further clearness and consistency in his thinking. He cannot do so with the human race, which nature has produced, constituting, from lowest to

highest, an unbroken continuum. All so-called types of human beings represent plus or minus variants, deviations of excess or deficiency, of the qualities and attributes inherent in all human beings.

Nature of the normal or Gaussian curve of distribution of traits. In studying human traits we are not only interested in knowing the size of the extreme measures and the fact that all other measures are intermediary, but we want to know the frequency of occurrence of each of the measures. How are

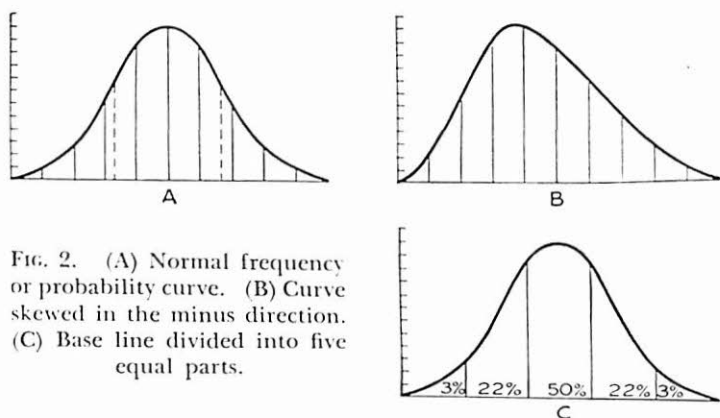


FIG. 2. (A) Normal frequency or probability curve. (B) Curve skewed in the minus direction. (C) Base line divided into five equal parts.

the measures distributed? Gauss formulated the mathematical law according to which the measures may be expected normally to distribute themselves. Such a "normal probability curve," or "normal curve of error," or "normal frequency curve or polygon," as it is also called, is shown in Fig. 2.

A perfectly normal curve, rarely found in practice in psychological, biological, or educational measurements, possesses the following fundamental characteristics.

(a) The measures will cluster around one "mode"—the measure of greatest frequency. The mode indicates the typical or central tendency of the determination. In a perfect unimodal curve the average, median, and mode coincide.

(b) Small variations from the central tendency are more frequent than large variations. The decrease is at first slow,

then rapid, and then slow again, in consequence of which the curve appears bell-shaped—hence the designation “bell-shaped curve.”

(c) The variations are continuous. There are no gaps or sharp division points between the different segments of the curve.

(d) The curve is symmetrical, that is, there are as many plus variations as minus variations.

FACTORS AFFECTING THE CURVE. These characteristics, however, apply only to the ideal curve. In practice the normal curve is only approximated. The closeness of the approximation may be augmented by increasing the number of measurements, by basing results on a chance distribution of cases, and possibly by confining the measurements to original traits as against acquired traits.

The number of measurements or observations of a continuous variable needed to yield a perfect approximation will vary with the type of material. For many traits (psychological, social, and educational) many thousands of measurements are required. The requirement of a chance array of traits is predicted on the assumption that the factors that make for minus and plus deviations from the mode will be equally potent or numerous in an unselected distribution. If the determinations are based on selected cases, the curve may be “skewed” toward the one side or the other, or it may contain two modes (bimodal) or several modes (multimodal).

Does the Gaussian curve apply to general intelligence? The distribution of general intelligence seems to approximate the normal probability curve, if the distribution is judged by the I. Q. curve in Fig. 3. This curve is based on Forms L and M of the “Revised Stanford-Binet Scale of Intelligence”²¹ administered to 2,904 representative or unselected American-born white children, ages two to eighteen, examined in a dozen states in the far west, middle west, south, and east, in rural and urban areas from different socio-economic levels.

²¹ Terman and Merrill, *Measuring Intelligence*, p. 37.

The curve, a reasonably close approximation, might have been perfect had 10,000 or 50,000 children been examined. However, it must be remembered that the tests were so located in the two scales as to "make the mean mental age of each group of subjects identical with the mean chronological age, giving a mean I. Q. as close as possible to 100."²² Moreover, whether the I. Q.'s from a wide range of ages based on dif-

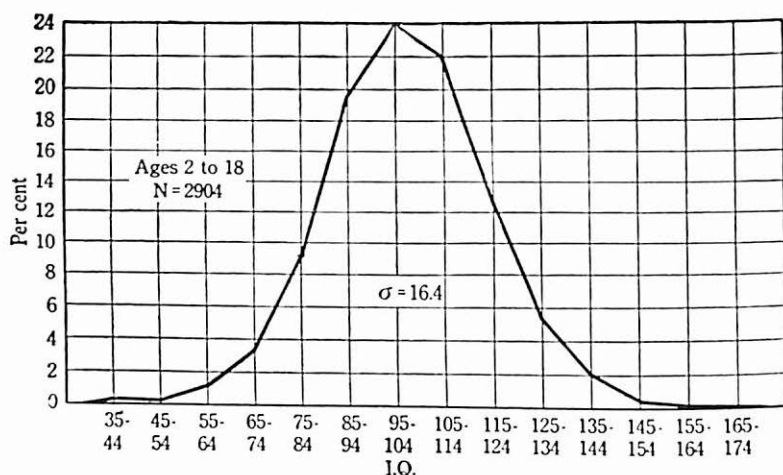


FIG. 3. Distribution of I.Q.'s based on the combined results of the Stanford-Binet Forms L and M.*

ferent kinds of intelligence tests can be legitimately combined into one curve of distribution is a question in need of investigation.

ADDITIONAL COMMENTS ON the normal curve as applied to intelligence. (a) The concept of a normal person as one who is perfectly symmetrically developed in all attributes is a myth. All persons possess "longs" or "shorts" in some degree.

(b) The concept of a subnormal or a supernormal person as uniformly or symmetrically deficient or talented in all directions is also a myth. Although there are some persons

²² Terman and Merrill, *Measuring Intelligence*, p. 21.

* From Terman, Lewis, and Merrill, Maud, *Measuring Intelligence*. Boston: Houghton Mifflin Company, 1937, p. 37. Reproduced with the publisher's permission.

who deviate more or less uniformly or generically, many are merely specifically disabled or gifted.

(c) There are temporary forms of subnormality or supernormality, although most persons probably continue to occupy their relative rank positions.

(d) Both subnormality and supernormality sometimes rest upon a neuropathic or pathological background. Of seven classes of bright children recognized in a Japanese study, most were classed as pathological or morbid. The belief is general that precocious children are delicate, puny, sickly, "all brain and no muscles," and doomed to early extinction, decadence, or insanity. Nevertheless, many supernormals are perfectly healthy and well-poised. The extensive Stanford University investigations of children with high I. Q.'s seem to show that the typical bright, precocious, talented child is healthier, mentally and physically, than the child of lower ability.²³

(e) Our concept of normality will differ according to race and social condition. It is idle to assume that norms for children in New York City would be applicable as measures of so-called native intelligence to those in Patagonia. The concept of "native intelligence" is, to a degree, also mythical.

(f) Extensive familiarity with the achievements of children in the schools has engendered the belief that the curve tends to be skewed toward the minus side, owing to the frequent action of the forces which tend to produce physical and mental deterioration: defective heredity, diseases, toxins, alcohol, tobacco, opiates, accidents, abuse, neglect, dissipation, indolence, and the like. The factors that make for deterioration are probably more numerous or potent than the factors that produce improvement, such as ambition, effort, exercise, education, hygiene, sanitation, eugenics, eugenics, and the like. It is easier to float with the current than to breast the rapids.

Meaning of mental or intelligence age in the light of the quantitative concept. A word of caution is pertinent at this point against drawing unwarranted implications from the

²³ Terman, Lewis M., *et al.*, *Genetic Studies of Genius*. Stanford: Stanford University Press, I, 1925; II, 1926; III, 1930.

psychometric classification of mental defectives. Thus, when we say that a child of twelve has a five-year mentality or intelligence level, all we mean is that he is approximately on a par with a normal five-year-old child as judged by the measuring scale employed, being inferior in some tests, and superior in others. We do not mean that he has the identical mental constellation or pattern of the five-year-old, or that his mental processes function exactly as they do in a normal child of five. The combination of traits may be quite disparate. The normal five-year-old would possess more initiative, spontaneity, naïveté, infantility, and potentiality for growth than the twelve-year-old with a five-year mentality. Moreover, the instinctive, emotional, and psychomotor development of the twelve-year defective would almost always be more mature or more highly advanced than in the normal five-year-old. He would perhaps be on the plane of the ten- or twelve-year-old in emotional development, and the seven- or eight-year-old in motor development. Twelve-year-old defectives of five-year intelligence level are not like kindergarten children and should not be trained like such pupils.

It would add to definiteness and clarity if the term "intelligence age" were abandoned for the term "test age," with an indication of the test used, such as Stanford-Binet Form L age, or Arthur performance age, or Wechsler-Bellevue age. The term mental age (M. A.) should not be employed as an equivalent of intelligence age (I. A.), as is ordinarily done, for mental age is a much broader concept than intelligence age, including, as it does, emotional, temperamental, social, motor, and intellectual elements and stages of maturity.

THE MENDELIAN UNIT-DEFECT VERSUS COMPLEX- DEFECT THEORY

*Is mental defectiveness a simple, unitary defect
or a complex condition?*

Some of the followers of Mendel, particularly during the first decade or two of the century, applied his theory of hered-

itary transmission to the explanation of mental defectiveness of the primary type. Mental defectiveness results, they held, from the absence of a unit trait or determiner in the germ cells (genes), the presence of which constitutes normal intelligence or normal-mindedness. Mental defectiveness behaves like a Mendelian recessive, and normality behaves like a dominant.

If this theory were correct, there should be a sharp cleavage between mental defectiveness and normality, and the problem of diagnosis would merely involve the determination of whether a particular individual does or does not possess the "unit trait" or "unit character" (normal-mindedness). If he does, he would be adjudged normal; if he does not, he would be classed as mentally defective. But, as we have already seen, such a sharp line of demarcation is purely imaginary. The distribution of intelligence is continuous from idiocy to genius. No gaps exist in the curve even between the mental defectives and those not mentally defective. Karl Pearson and G. A. Jaederholm found on the basis of Binet tests that "70.5 per cent of normal children fall into the range of intelligence of the so-called mentally defective; and 60.5 per cent of so-called mentally defective children have an intelligence comparable with that of some normal children." The large amount of overlapping found here was probably due to the presence of backward children among the mentally defective and dullards among the normals. But that circumstance does not invalidate the main argument. Discrepancies in diagnosis are caused by the fact that there is no split in the distribution. This fact constitutes a strong presumption in favor of the rival theory.

Complexity of mental defectiveness. From these and other considerations we reach the conclusion that mental defectiveness, instead of being a simple classifiable entity due to a constant specific cause with definite restricted and invariable symptoms, such as diphtheria or typhoid fever, is a highly complex condition. It is complex in respect to the fact that

(1) it may affect in unequal degree various physical, intellectual, volitional, temperamental, emotional, social, and moral functions; (2) it may cover a wide range of defect from profound idiocy to high grade mental defectiveness; (3) it may be caused by a great variety of etiological factors, hereditary and acquired; and (4) the underlying brain defect (pathology) may differ greatly in different cases.

Individual differences among the mentally defective. Let us remember that there are pronounced differences in physique, disposition, attitude, emotional attributes, energy, application, and social and moral traits between mentally defective children even of the same grade of intelligence or degree of impairment, just as there are similarly pronounced differences between normal children of the same age, maturity, or intelligence level. No two idiots are exactly alike: some are apathetic, others excitable; some have more of one trait, others more of another. The "typical imbecile face" of established tradition is largely mythical. Some imbecile faces are beautiful, others ugly and repulsive; some look intelligent, others vacuous. Some imbeciles are mobile, others immobile; some are well-behaved, others are great trouble-makers. Few characterizations apply to the whole class of the mentally defective, and fewer still to every mentally defective individual. Between mental defectives, as between normal individuals, diversity, unevenness, and variation are the rule, not uniformity, symmetry, and sameness. Nature seems to prefer variety. What a drab, boring existence life would be if all humans, even subnormals, were alike! It follows that descriptions of mental defectives must be largely restricted to generalizations respecting the average or most frequent representatives of the different grades or physical types. Owing to the demonstrated existence of individual differences in the mode of origin (pathogenesis), in the underlying brain defect (pathology), and in the manifestations (symptomatology), it is obvious that there can be no simple, single, or infallible test of mental defectiveness such as would be demanded by

the assumption that the defect is due to the absence of a unit trait.

Let us discard the mistaken view that mentally defective children are radically different from normal children—and this is particularly true of the higher grades—and should therefore be treated as outcasts devoid of all human rights, as was the case in ancient times when they were called idiots (*idios*, one's own, or extra-social), and were treated with scorn and aversion, or were abandoned in the mountains to die from starvation or wild animals, or were thrown in rivers to perish from drowning.

Chapter 4

THE RELATIONSHIP BETWEEN MENTAL DEFICIENCY AND MENTAL DISEASE

The prevalence of psychosis (mental disorders or disease)¹ among intellectually normal and subnormal children

An undetermined number of intellectually normal and subnormal children are mentally disordered. The exact number is not known for obvious reasons.

The borderline between normality and abnormality is a broad, ill-defined, and uncertain zone, with respect to both mental defect and mental disorder. Psychotic children are often confined in a great variety of institutions, such as hospitals for the insane, institutions or colonies for the feeble-minded, correctional institutions, orphanages, or other kinds of child care institutions. Some psychotics, even in institutions, may have remained unrecognized and unreported.

Many psychopathic, psychotic, and pre-psychotic children remain at home or are at large in society. No psychiatric and psychological surveys have ever been made of all the children in any considerable population group to determine the size of the juvenile psychiatric group. The available statistical findings are based on selected cases, usually institutional commitments, and the statistics for the population at large are mere estimates. Louttit, after reviewing a number of studies, quotes with apparent approval the opinion of Moeller that 25 times as many psychiatric children are at large in society

¹The popular term for a psychosis is insanity. This is also the term employed in legal parlance after the individual has been legally adjudged insane.

as are found in hospitals.² Whether this estimate made in 1876 would hold now is problematical.

Generally, it is more difficult to recognize psychoses in children than in adults because some of the obtrusive signs of mental disorder are less patent in children, such as deterioration of judgment, loss of insight, sudden change of personality, perceptual disturbances, illusions, hallucinations, and delusions. Affective disorders may be more difficult to interpret. The writer has examined a number of adolescents diagnosed as cases of dementia praecox who were suffering from temporary emotional disturbances and who eventually proved to be normal and not psychotic. Doubtless many children, both intellectually normal and feeble-minded, have been mistakenly classified as psychotic.

Of 74,985 "first admissions with psychosis" to state hospitals for mental disease in 1944, less than .5 per cent (357) were under fifteen years of age, and about 4.4 per cent were between fifteen and nineteen, making somewhat less than 5 per cent under twenty.³

Opinion regarding the prevalence of psychosis among mental defectives has been quite divergent. Some authorities have held that the mentally defective possess a certain immunity against acquiring mental disorders and that the acquisition of a mental disease presupposes a considerable level of mental integration. Thus Bartemeier maintains that few psychotics are found among mental defectives because "their relatively simpler nervous system renders them less sensitive to disturbing influences than is the normal individual."⁴ Others maintain that mental defectives, because of their innate instability and lack of neuronie resistance, lose their

² Louttit, *Clinical Psychology*, p. 532.

For a brief review of the American writings between 1812 and 1898 on child mental disease see Rubinstein, E. A., "Childhood Mental Disease in America," *American Journal of Orthopsychiatry*, April, 1948, 314-321.

³ Advance statistics for mental hospitals from the U. S. Bureau of the Census, August 7, 1946.

⁴ Bartemeier, Leo H., "Psychoses in the Feeble-Minded," *Journal of Psycho-Asthenics*, 1925.

mental equilibrium more easily under the influence of alcohol and the excitements, worries, and stresses of social and economic adjustments. Consonant with this view, Berkley maintains that morons are more prone to mental disorders than normal persons,⁵ and Tredgold finds the incidence of psychoses 26 times greater among mental defectives than in the general population.⁶ The more recent estimate of Neuer is that "at least 20 per cent of patients who function on the level of a high grade mental defective manifest signs of a minor or major psychosis."⁷ The most frequent type of psychosis among mental defectives is the manic-depressive disorder, according to Tredgold, and schizophrenia (also called dementia praecox), according to S. I. James. Rollin classified all of his psychotic mongols as cases of "primitive catatonic psychosis" (p. 291).

The ratio of psychotic mental defectives contained in statistics from institutions for the psychotic and the mentally defective are considerably lower. Thus, only 3.6 per cent of all first admissions to the Massachusetts hospitals for mental disease in 1936 were diagnosed as "psychoses with mental deficiency," and only 7.5 per cent of all the hospital inmates that year were so diagnosed.⁸ In the Danville, Pennsylvania,

⁵ Berkley, Henry J., "The Psychoses of the High Imbecile," *American Journal of Insanity*, October, 1915, 305-314.

⁶ Quoted from Kallmann, Franz J., Barrera, S. Eugene, Hoch, Paul H., and Kelley, Douglas M., "The Role of Mental Deficiency in the Incidence of Schizophrenia," *American Journal of Mental Deficiency*, 1941, 514-539. In the sixth edition of his *Mental Deficiency* (p. 380), Tredgold says merely that "mental disturbance or decay is very much greater in defectives than in non-defectives." See also Pollock, Horatio M., "Mental Disease Among Mental Defectives," *American Journal of Mental Deficiency*, April, 1945, 477-480; Feldman, Fred, "Psychoneuroses in the Mentally Retarded," *American Journal of Mental Deficiency*, October, 1946, 247-254. Rohan, J. C., "Mental Disorder in the Adult Defective," *Journal of Mental Science*, 1946, 92: 551-563. Weaver, Thomas R., "The Incidence of Maladjustment Among Mental Defectives in Military Environment," *American Journal of Mental Deficiency*, October, 1946, 238-246.

⁷ Neuer, Hans, "Prevention of Mental Deficiency," *American Journal of Mental Deficiency*, April, 1947, 727.

⁸ Pearson, Grosvenor B., "The Psychoses with Mental Deficiency as Viewed in a Mental Hospital: Clinical Syndromes," *Journal of Psycho-Asthenics*, 1938, 1: 166-172.

State Hospital, in about 1930, 6.3 per cent of the patients fell into the same category.⁹ It is recognized, of course, that some of the psychotic mental defectives were in the institutions for the mentally defective. Between 1920 and 1930 in the Walter E. Fernald State School for the feeble-minded in Massachusetts 12.4 per cent of the inmates "showed evidence of psychosis" (Greene).

The discrepancies in the views of different physicians are caused by a number of factors.

(1) The distinction between a mental defect and a mental disorder, such as the simple hebephrenic type of dementia praecox or the rare conditions called "precocious dementia" (by S. De Sanctis in 1906) and "infantile dementia" (by Theodore Heller in 1909),¹⁰ is not always clear-cut. This causes confusion and disagreement in diagnosis. Schizophrenics of the simple type have often been sent to institutions for the feeble-minded under an erroneous diagnosis, where they may have remained for years before a correct diagnosis was made. They have also been entered in special public school classes, although this happens less frequently because most of these classes deal with younger children. Some of the personality deviates sent to these classes as mental defectives are probably pre-schizophrenics.

(2) It is easy to misjudge the intelligence level of psychotics when the estimation is based on clinical observation unaided by test findings. Some psychotics classified even as idiots or imbeciles may be fairly normal intellectually as determined by tests.¹¹ How many diagnosed as feeble-minded are cases of pseudo-feeble-mindedness rather than genuine feeble-mindedness cannot be determined.

(3) Many of the feeble-minded are occasionally subject to

⁹ Quoted from Greene, Ransom A., "Psychoses and Mental Deficiencies, Comparisons and Relationship," *Journal of Psycho-Asthenics*, 1930, 128-147.

¹⁰ See Louttit, *Clinical Psychology*, p. 515; Penrose, Lionel S., *Mental Defect*, New York: Farrar & Rinehart, Inc., 1931, p. 140.

¹¹ On this point consult Wallin, *Problems of Subnormality*, Yonkers: World Book Company, 1917, pp. 157-165.

psychotic or psychoneurotic episodes, usually transitory in character (although they may last for days), which may resemble almost any symptoms found in any of the psychopathic groups, such as periods of excitement, emotional outbursts, maniacal attacks, agitated or passive depression, anxiety, apathy, indifference, resistance, obstinacy, irritability, seclusiveness, withdrawal from social contacts, unprovoked laughter or crying, hysterical pains or paralyses, childish behavior, night terrors, specific phobias, confusion, delusions, hallucinations, automatisms (such as meaningless repetition of words or actions), quaint mannerisms, and the like. These abnormal behavior patterns essentially represent faulty reaction patterns that the subject adopts, consciously or unconsciously, for solving his difficulties of adjustment. The particular pattern adopted depends upon the situational factors and the personality make-up and intelligence level of the individual. Because of the extensive overlapping between some members of the two groups and the great variety of the psychotic phenomena found among both high grade and low grade defectives,¹² they may be differently classified by different examiners either as mental defectives of the unstable type or with personality disturbances, or as psychotic or psychopathic mental defectives, or as psychotics with mental deficiency.

(4) The estimates may have been based on different age groups. The younger groups will yield fewer psychotics than the older groups.

Nature of causal relationship between mental defect and mental disorder

Widely divergent postulates have been advanced regarding the causal relationship between the endogenous form of men-

¹² For a brief delineation of the differences seen in the psychotic manifestations of idiots, imbeciles, and morons see Pearson, G. B., "The Psychoses with Mental Deficiency as Viewed in a Mental Hospital: Clinical Syndromes." Illustrative cases are found in this reference and also in Whitten, B. O., "Psychotic Manifestations of Mental Defectives," *Journal of Psycho-Asthenics*, 1938, 1: 72-79; Greene, "Psychoses and Mental Deficiencies," p. 136.

tal deficiency and idiopathic mental disease. The following are some of the discordant views on the causal relationship between mental deficiency and schizophrenia. (1) Both conditions have the same etiological background; they originate from the same genotype (fundamental hereditary combination of genes). (2) Both are the product of a common process of "decerebration," produced by genetically determined increased vulnerability of the brain to injuries at birth or in early childhood. (3) Schizophrenia is grafted upon feeble-mindedness. (4) Mental deficiency modifies schizophrenia and schizophrenia reinforces mental deficiency. (5) Mental deficiency in the ancestry predisposes to schizophrenia. (6) There is no etiological connection between them. (7) Although originating independently, they influence one another. (8) The occasional concurrence is purely accidental or coincidental.

In one of the most recent studies of the etiological relationship of mental deficiency and schizophrenia, based particularly on 365 pairs of twins, with "schizophrenia or mental deficiency in one or both members of a set," Kallmann, Barrera, Hoch, and Kelley reach the conclusion that "the concurrence of schizophrenia and mental deficiency has been merely coincidental at least in those twin cases showing the idiopathic form of either trait."¹³ They did not find "one single pair of one-egg twins in which one member was definitely schizophrenic and of normal intelligence, and the second member was mentally defective and free of schizophrenia." They failed to find any "evidence either of an increased incidence of schizophrenic psychoses in the consanguinity of mental defectives or of an increased tendency for the blood relatives of schizophrenics to be feeble-minded. . . . The endogenous forms of schizophrenia and mental deficiency are based on different genetic factors which are

¹³ Kallmann, *et al.*, "Role of Mental Deficiency in the Incidence of Schizophrenia," pp. 522, 523. See the studies also cited by Myerson, Abraham, *et al.*, *Eugenical Sterilization: A Reorientation of the Problem*. New York: The Macmillan Company, 1936, pp. 90 f.

specific and not related to each other. . . . In practically all of our mentally defective twin pairs with discordance for a psychosis, it is the less intellectually inferior twin who has been found to be psychotic."¹⁴ Neuer's sweeping conclusion that "the mental deficiency in neurotic or psychotic children is . . . the result and not the underlying cause of a minor or major psychosis in childhood" is not based upon the presentation of unambiguous data that can be interpreted in only one way—in fact, the statement is made without any documentation.¹⁵ The time is not ripe for pronouncing the final verdict on this intricate question.

Methods of differentiating mentally deficient and psychotic children

Psychotic and congenitally mentally defective children can frequently be differentiated with considerable accuracy on the basis of the case history. The congenitally defective child shows evidence of his slow rate of development and limited ability from early life, although his deficiency may not be particularly noticeable until he is confronted with the standardized learning situations of the school environment. Impaired ability to learn abstract subject matter and to adjust to novel situations are two of his outstanding defects. In addition to a retarded rate of development, premature arrest of development also occurs in some cases. The psychotic child, on the other hand, may be perfectly normal intellectually in early life and may grow and develop uneventfully at a normal rate until some crisis occurs—a development arrest or perversion or cerebral trauma—when he begins to manifest bizarre behavior, such as loss of interest in his surroundings and in his comrades, mental stagnation or deterioration, failure in studies, oddities of behavior, misconduct, intro-

¹⁴ Kallmann, *et al.*, "Role of Mental Deficiency in the Incidence of Schizophrenia," p. 37.

¹⁵ Neuer, "The Relationship Between Behavior Disorders in Children and the Syndrome of Mental Deficiency," *American Journal of Mental Deficiency*, October, 1947, 147.

version, seclusiveness, unsociableness, excess emotionality, and the like. The differentiation of the more subtle cases requires the services of a skilled psychiatrist or clinical psychologist, and the aid of psychological and other test techniques.¹⁶

The function of the classroom teacher and the educationist is to familiarize themselves with the signs and symptoms of mental and nervous disturbances in children and the symptom complexes in juvenile psychotics and pre-psychotics and to refer suspicious cases to experts in mental diagnosis. Teachers, who are among the first public officials to contact the general run of children intimately, should be sensitized to a due appreciation of the importance of discovering and treating mental disorders in their incipency when they can be more easily adjusted. The main symptoms of the major form of juvenile psychosis are briefly described in the following pages.¹⁷

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Schizophrenia

Incidence. Schizophrenia is the most prevalent type of both the adult and the juvenile form of psychosis. In 1944, 22.6 per cent of the first admissions of psychotics to "hospitals for the permanent care of psychiatric patients" were classified as schizophrenics as compared with 9.2 per cent of manic-

¹⁶ Kallmann, *et al.*, "Role of Mental Deficiency in the Incidence of Schizophrenia," pp. 532 f.

¹⁷ For a brief statement on the symptoms of child neuroses see Alpert, Augusta, "Criteria for Recognition of Neuroses in Children: A Guide for the Pediatrician," *American Journal of Diseases of Children*, September, 1946, 274-278.

depressives, the next most prevalent type of functional cases. Of the first admissions "with psychosis" to state hospitals during the same year, 28.5 per cent of those under fifteen years of age (357 cases) and 54.6 per cent of those under twenty (3,306 cases) were diagnosed as schizophrenics.¹⁸

About 60 per cent of praecox cases develop before the age of twenty-five, some as early as five years, according to one authority. Obviously, this is the type of mental disorder which affects both sexes with only a slight preponderance among males,¹⁹ with which persons dealing with youths should be peculiarly concerned. Only the highlights of this protean disorder can be reviewed. In all its complicated forms it is, perhaps, the most curious and bizarre of the psychoses.

Terminology. Various terms have been applied to the disorder, such as adolescent insanity, by Thomas S. Clouston; dementia praecox (early), by Emil Kraepelin in 1896; and schizophrenia (*skhizein*, to split or divide; *phren*, mind), by Eugen Bleuler in 1911. The latter term has largely displaced the once-popular dementia praecox, because the disease does not always lead to dementia, because it often occurs in late adulthood (the so-called delayed form), and because the new term describes one of the basic characteristics of the disorder—the splitting asunder of the psyche into disconnected fragments. This disharmony or lack of coordination (also referred to as intrapsychic ataxia by Stransky) between the patient's emotions, thoughts, and conduct is offered as the explanation of his irrational, ludicrous, and grotesque behavior.

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¹⁸ Bureau of Census Report, August 7, 1946.

¹⁹ Twenty-two per cent males and 19.7 per cent females, based on first admissions to hospitals for permanent care in 1943: *Patients in Mental Institutions, 1943*. Washington: Bureau of the Census, Government Printing Office, 1946, p. 22.

normal Psychology. Baltimore: The Williams & Wilkins Company, 1935, pp. 224-240.

Louttit, Chauncey, *Clinical Psychology*, pp. 542 ff.

Scheidemann, Norma T., *Psychology of Exceptional Children*. Boston: Houghton Mifflin Company, 1937, II, pp. 265-268, 280-290.

Major characteristics. The disorder cannot be adequately characterized in a few paragraphs because of its multiform manifestations and the marked differences between the four major forms—the simple, the hebephrenic, the paranoid, and the catatonic—and the great individual variations that occur according to the severity and stage of the malady and the intermixture of the four forms. Some symptoms are common to all types and cases; others are typical of only certain forms or stages. Some of the more common characteristics are listed below.

(1) Emotional vacuity, blunting, or indifference, manifested in lack of ambition, loss of interest in work, surroundings, family, and friends; apathy and idleness; seclusiveness, “shut-in-ness,” preoccupation with inner thoughts and feelings, withdrawal from outside realities (“flight from reality,” sometimes with the incapacity to distinguish between fact and fancy); indifference to personal appearance, slovenliness, untidiness, and loss of pride. In extreme cases he may, apparently, become completely oblivious of his surroundings and wholly inaccessible, refusing to answer questions or respond to requests.

(2) Incongruity between affect (feelings), ideation, and behavioral reactions, shown in such inappropriate acts as crying upon the receipt of good news, or laughing over tragic events, or feeling amused by events that should cause serious concern. Instead of such contrary reactions the responses may be inadequate. He may merely smile or simper when he should laugh, or respond languidly to stimuli that ordinarily provoke anger or affection, as the case may be.

(3) Partial intellectual dilapidation, which may manifest

itself in a variety of ways, such as: intellectual deterioration (often rapid and profound in hebephrenics); inability to concentrate; interludes of confusion; peculiar, fantastic, silly ideas, or delusions, often transitory, involving ideas of grandeur, suspiciousness, persecution, ideas of reference (that he is the object of unfavorable comment), and thoughts about being influenced (by electrical currents passing through the body, by radioactive substances, noxious odors, or mysterious forces); transitory hallucinations, often unsystematized (usually auditory or visual and disagreeable); and incoherent or irrelevant speech, sometimes referred to as "word-salad" (a flow of words without any logical connection). The patient may invent new words (neologisms) to express his strange feelings. Judgment and insight are almost always impaired.

To illustrate: "The patient hears mocking, accusing, or threatening voices, or sees menacing gestures, hideous monsters, or fiery devils (disagreeable hallucinations). She is convinced that she has committed the unpardonable sin or suffers from an incurable malady, such as cancer, syphilis, or brain softening. She believes that the alimentary canal has been closed, that she has no brain, eyes, or hands, or that the viscera have been turned into glass or wood. She is tormented by the belief that she is impotent, vile, and forever damned (depressive delusions), or that people hate her, are trying to poison, seduce, or murder her (persecutory). She is exhilarated by the thought that she is the Holy Ghost, Queen of the Universe, the mother of God, enormously affluent, lives in a palace, or wears diamonds (grandiose delusions). The patient usually can give no explanation for such evident absurdities of thought or of conduct."²⁰

(4) Queer, ridiculous, or freakish behavior exhibited in a great variety of ways, such as impulsive outbreaks (often produced by hallucinations or delusions); repetition of meaningless phrases (language stereotypy); automatic repetition

²⁰ Wallin, *Clinical and Abnormal Psychology*, p. 285.

of questions or remarks uttered by others (echolalia); automatic repetition of movements (stereotypy of movements or autopraxia); grimacing; assumption of odd postures or other mannerisms, attitudinizing or stereotypy of attitude (sitting in a certain position on a certain bench, keeping the eyes fixed on a corner of the ceiling, talking, walking, or eating in a peculiar manner); retention of limbs in any positions in which they have been placed (flexibilitas cerea); negativism (doing the opposite of what is wanted or refusing to do anything, such as answering questions, obeying the calls of nature, dressing, or eating); tensing the muscles (closing the mouth or eyes tightly or clenching the fists); catatonic stupor, in which the patient may lie rigidly motionless for hours but retain consciousness of what is transpiring; and mutism.

The more bizarre behavior patterns, which may come and go, often unpredictably, and which may represent some form of escape or defense mechanism that the patient adopts, consciously or unconsciously, are not found in the simple type. Some pertain only to the hebephrenic type, and others to the catatonic. Many symptoms occur in the mixed type. No patient shows all the symptoms. The disorder ordinarily develops slowly and insidiously, but may occur abruptly in connection with some crisis of development, such as an acute illness, an emotional shock, a severe fright, alcoholic excesses, worry over sex problems or other problems, menstrual disorders, childbirth, head injuries, and subjection to a new difficult pattern of living or of work.

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Shakow, David, "The Nature of Deterioration in Schizophrenic Conditions," *Nervous and Mental Disease Monographs*, No. 70. New York: Coolidge Foundation, 1947.

Wallin, J. E. W., *Problems of Subnormality*, pp. 157-165 (one of the first applications of the Binet test to psychotics).

For a detailed differentiation between the brain injured and schizophrenia, encephalitis, and psychopathic and neurotic manifestations, see Strauss, Alfred A., and Lehtinen, Laura E., *Psychopathology and Education of the Brain-Injured Child*, pp. 87-97.

Theories of causation. Complete agreement has not yet been reached on the basic cause and the relative importance of putative contributory or precipitating factors of schizophrenia. The two hypotheses most widely held are the following. (1) The theory of neuropathic inheritance; some psychiatrists have affirmed that schizophrenia is the most hereditary of all psychoses. Thus Wolfson found an hereditary taint in 90 per cent of his cases.²¹ (2) The psychogenic theory (or the psychobiological theory of Adolph Meyer, its chief early protagonist). This theory ascribes a dominant role to the failure of the individual to adjust successfully to

²¹ Louttit, *Clinical Psychology*, 1936, p. 533.

the social demands of the environment,²² and the resultant development of serious mental conflicts, frustrations, and maladjustments and the accumulation of faulty habit patterns and unwholesome methods of sidestepping difficulties by the adoption of some form of escape mechanism, such as regression to lower levels of adjustment (as in hebephrenia), obtaining satisfaction in the world of fantasy (as in the paranoid delusions of grandeur), detachment from the grim world of reality (as in periods of unconsciousness, stupor, or shut-in-ness).

The collateral assumption is usually that a schizoid personality make-up constitutes fertile soil for the development of schizophrenia, although not all schizoid children become mentally diseased. The term schizoid is applied to the shy, shut-in, unsocial, introspective type of child who cannot adapt himself to the conditions of life or cope with his studies and therefore tends to withdraw from external contacts and to spend his time in introvert ruminations and brooding. The schizoid child tends to be conscientious, sensitive to criticism, limited in his interests, and more interested in abstract than in concrete ideas. He prefers solitary amusements and occupations to group play or activities. He is inclined to become irritated and resentful when his fantasy (autistic) thinking is interrupted. The personality change is related to a disorder of affect, a deterioration in disposition and mood.

General agreement is lacking on many other reported causative factors, such as glandular disorders (autointoxication from sex gland dysfunctioning), disturbances of protein

²² Sometimes a perverse home environment increases the hazards to the child for the attainment of mental health and stability. The turmoil created by psychotic, criminalistic, defective, abusive parents or parents who do not afford normal parent-child relationships is vividly presented in Bender, Lauretta, "Behavior Problems in Children of Psychotic and Criminal Parents," *Genetic Psychology Monographs*, 1937, 19: 229-339. See also the following investigation and suggestions derived therefrom, Yerburg, Edgar C., and Newell, Nancy, "Factors in the Early Behavior of Psychotic Children as Related to Their Subsequent Mental Disorder," *American Journal of Mental Deficiency*, July, 1942, 70-76. For an evaluation of the hereditary data, see Myerson, *et al.*, *Eugenic Sterilization*, pp. 90-107.

or oxygen metabolism, cortical degeneration (sclerosis, fatty degeneration, cell atrophy, and lipid changes in many areas of the brain), impaired circulation (from a small heart), and an asthenic body build. The asthenic type (literally, weak), according to Emil Kretschmer, has long limbs, a long, flat chest, a thin neck, and poor muscular development.

Preventive and curative treatment. The preventive problem is to discover the excessively introverted, shut-in, diffident, unsocial child and to develop his confidence in himself, overcome his egocentricity, and develop his gregarious and altruistic impulses. The process is one of objectification of attention, socialization, and activation instead of fruitless meditation. The goal is to divert the child's attention from himself to wholesome outside interests and activities; get him interested in objective realities, in the workaday world, in personal and social accomplishments that win the approval of pals, parents, and teachers, and in the activities of other children and adults. Develop healthy objective-mindedness in place of morbid subjective preoccupation and rumination. Train him to meet his difficulties objectively and unemotionally. Provide opportunities on the objective reality plane that will afford satisfying outlets for his energies. Satisfying constructive achievements in work and play and active participation in the activities of other children and adults will tend to keep his mind off himself and will yield greater joy and satisfaction than the temporary illusory delights that may spring from indulgence in daydreaming. Such activities will prove healthful and may serve as a corrective of the disruptive schizoid tendencies that may culminate in schizophrenia. The problem of the education and social care of the shy, quiet, inhibited, solitary, ruminant child requires the skilled application of mental hygiene and psychotherapeutic procedures, in addition to such supportive medical treatment as may be indicated.²³

²³ For further preventive and corrective suggestions, see Wallin, J. E. W., *Personality Maladjustments and Mental Hygiene*, New York: McGraw-Hill Book Company, Inc., 1935, especially pp. 318-322, 315-358.

The prognosis for those who have already become schizophrenic is more favorable under modern treatment procedures than used to be the case. A certain number of schizophrenics recover spontaneously. A larger number recover or are greatly improved by the modern combination of re-education, habit training, socialization, physiotherapy (including hydrotherapy or sedatives for conditions of excitement and massage for contractures), diversified occupational therapy (both intellectual and manumental), and supportive and specialized medical care. Of the 17,158 schizophrenics discharged from state hospitals in 1943 (the largest number of discharges of any type of psychotics), 19.4 per cent were recorded as "recovered," 67.5 per cent as "improved," and 11.8 per cent as unimproved.²⁴

SHOCK TREATMENT. The highly specialized forms of medical treatment include some form of shock treatment for throwing the patient into a coma or state of unconsciousness, with or without convulsions. The earliest form of shock treatment, the so-called "sugar shock" treatment, was introduced by Manfred Sakel, Viennese physician, in 1928. The treatment consists in giving daily injections of insulin in progressive doses until the sugar in the blood is very low. (called hypoglycemia). Then a massive dose is administered which throws the patient into a deep coma after a few hours. The return of consciousness is brought about by feeding the patient sugar (glucose). Upon the return of consciousness the mental condition often clears up and mental reintegration is established.

Many other methods have been employed to shock the patient into unconsciousness, such as the use of camphor, the inhalation of nitrogen, the injection of metrazol (a treatment devised by Ladislaus von Meduna of Budapest in 1934), and the shooting of an electric current of less than 100 volts through the head. A sufficient amount of metrazol is injected to produce epileptiform convulsions and unconscious-

²⁴ *Patients in Mental Institutions, 1943*, Bureau of the Census, 1946.

ness. The injections are usually repeated 10 to 15 times. The treatment is rather drastic and is often preceded by the use of curare to lessen the violence of the spasms. Certain types of schizophrenics (the catatonic and paranoid forms) are said to respond better to the insulin than to the metrazol. The electric shock treatment, introduced in its modern form by Cerletti and Bini in Rome in 1938, has superseded the chemical methods in many places because it is milder (especially in the form of electroshock therapy), more easily controllable, largely harmless, and apparently equally potent, although it causes more memory loss after treatment than metrazol. Usually from 10 to 20 treatments are administered at intervals of two or three times weekly. It is being applied to other forms of mental disease, especially those characterized by depressions, such as manic-depressive psychosis and the psychoses incident to old age and the menopause. According to Kolb and Vogel, 94 per cent of 305 state mental hospitals have used some form of shock therapy.²⁵ The reports vary from glowing accounts of spectacular cures to records of moderate improvement or failures.

To cite two reports on the use of insulin issued in May, 1944: 60 per cent of 316 patients in the Trenton, New Jersey State Hospital treated since 1939 "had shown definite improvement." The results obtained during a seven-year period with 700 patients in the Central Islip, New York, State Hospital were "not as effective as reports have indicated." Nevertheless, worth-while results were obtained with cases up to two years' standing. It is usually conceded that the shorter the duration of the malady, the more effective is the treatment. The various forms of shock therapy seem to be ineffective with children. The patients must be in good physical condition to withstand the shock ordeal.²⁶

²⁵ Kolb, Lawrence, and Vogel, Victor H., "Use of Shock Therapy in 305 Mental Hospitals," *American Journal of Psychiatry*, July, 1942, 90-100.

²⁶ The Committee on Therapy of the Group for the Advancement of Psychiatry has recently warned against the indiscriminate use of electroshock treatment.

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PSYCHOSURGERY is the term ordinarily used in connection with the form of prefrontal lobotomy (cutting into the lobe)

introduced by Egas Moniz in Portugal in 1936. The operation consists in severing bilaterally the subcortical fibers (white nerves) that connect the frontal lobes with the thalamus, a mass of gray nerve tissue at the base of the brain, which is supposed to be functionally connected with the emotions—a sort of physiological battery for “charging” ideas with emotional contents. No part of the brain is removed and the cortical areas are left intact. The implication is that the break in the neural connection between the intellectual and emotional life reduces the emotivity or excitability of the subject and renders him less apprehensive, fearsome, and impulsive.

Psychosurgery (see page 259) has been applied to intellectually normal persons subject to worries, depressions, fears, phobias, doubts, and delusions and to different kinds of psychotics, with varying immediate results, often unfavorable. The unfavorable results include confusion, restlessness, forgetfulness, loss of initiative and of planning capacity, and psychomotor retardation. Varying long-range favorable results have included relief from anxieties, fears, inhibitions, and excessive impulsiveness. Varying results have also been obtained with psychotics. One report issued in May, 1944 states that the operation enabled 50 per cent of schizophrenics to “lead useful lives.” This procedure invites searching analytical and statistical validation on a large scale over a period of years.

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Chapter 5

THE DEFINITION OF THE BACKWARD CHILD— THE DULLARD OR DULL NORMAL

The large group of children now to be considered, although not mentally defective in either the intellectual or the social sense, are genuinely lacking in all-around mental capacity or ability, varying from a level practically indistinguishable from high grade mental defectiveness to a level almost indistinguishable from normality. They are misfits in the regular grades in school. These children have been indifferently referred to as backward or retarded children, or laggards, dullards, or dull normals.

Characteristics in school. Reports which the author has received from the teachers of thousands of such children indicate that they tend to be listless and inattentive, rapidly lose interest in the regular school work, and fatigue easily, or rather grow weary rapidly—obviously because of lack of interest in the subject matter which is frequently incomprehensible and does not appeal to them. They are often prone to attacks of stubbornness, irritability, and bad temper, and suffer from a certain degree of moral obliquity or obtuseness. Backward children, more frequently than the mentally defective, are the disciplinary bugbears of the teacher and frequently of the other pupils, especially the smaller ones, whom they tease and bully.

Conduct disorders among backward children

In Pearson's study of intelligent, dull, robust, and delicate children among the English professional classes, it was found

that the intelligent children were far more conscientious and far less surly, and usually possessed greater athletic power and more robustness than the dull ones, whereas the delicate ones were below the average in intelligence. He concluded that there was a close connection between intelligence, morality, and physical robustness.¹

As a result of the clinical studies of delinquent and troublesome children in the St. Louis schools, the writer concluded that "it is particularly the backward pupil who creates the problems of discipline in the schools," who is "a more aggressive and intelligent troublemaker, and constitutes potentially a greater criminal menace" than the mentally defective child. Only 10.8 per cent of boys admitted in rotation to a class for truant, refractory, or incorrigible boys were diagnosed as mentally defective compared with 65 per cent who were diagnosed as dullards (borderline and backward grades of intelligence). Of 444 children with a record of misconduct, usually of a less grave form, 24.3 per cent were diagnosed as mentally defective and 62.4 per cent as borderline and backward, as compared with 35.7 per cent mentally defective and 53.3 per cent borderline and backward for the entire block of 2,774 consecutive cases, most of them reported because of suspected mental deficiency or inferiority. (See Table V.)²

A similar conclusion was later announced by Porteus after an investigation of inmates in an institution for the feeble-minded. Of 377 classified as feeble-minded (all with an I. Q. below 75), 64 per cent were well adjusted, and only 13 per cent had delinquent tendencies. Only 6.3 per cent of those with I. Q.'s below 55 were delinquent. On the other hand, only 33.3 per cent of 87 dull normals were well adjusted, and

¹ Pearson, Karl, "On the Relationship of Intelligence to Size and Shape of Head, and to Other Physical and Mental Characters," *Biometrika*, October, 1906, 105-116. For an extensive investigation of these and other relationships in bright children see Terman, Lewis M., *The Gifted Child Grows Up*. Stanford: Stanford University Press, 1917.

² Wallin, *Problems of Subnormality*, pp. 248 f.; and "Feeble-Mindedness and Delinquency," *Mental Hygiene*, October, 1917, 585-590.

49.4 per cent showed delinquent tendencies. "This shows that it takes a certain amount of intelligence to get into serious mischief."³

The writings on the relation of mental defectiveness and intelligence to delinquency and crime are entirely too extensive to be summarized here. Suffice it to say that all authorities are now agreed that in the early days of intelligence testing a wholly exaggerated emphasis was placed on the role of feeble-mindedness and low intelligence in the production of delinquent and criminal tendencies and behavior disorders.

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³ Porteus, Stanley D., *Studies in Mental Deviations*. Vineland, N. J.: The Training School Publications, No. 24, 1922, pp. 21 f.

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The growing conservatism on this question is shown by the following percentages of feeble-mindedness among delinquents and criminals reported in successive decades, based on the medians of many investigations compiled by Sutherland.⁴

For 1910-1914, 51 per cent (based on 50 studies)

For 1915-1919, 28 per cent (142 studies)

For 1920-1924, 21 per cent (104 studies)

For 1925-1928, 20 per cent (46 studies)

From their reviews of the material the conclusions were reached by Zeleny that 1.8 criminals exist for every non-criminal with an intelligence age less than eleven, and by Metfessel and Lovell that in the main the studies place the typical delinquent in the dull normal class, a conclusion reached by the author in the early part of the second decade of the century.⁵

Misbehavior and vocational incompetency due to maladjusted curriculum. Undoubtedly much of the misbehavior of backward children in school is directly attributable to their lack of interest in the regular curriculum and their inability to meet the ordinary requirements. Frequently when such

⁴ Sutherland, Edwin H., *Principles of Criminology* (3d edition). Philadelphia: J. B. Lippincott Company, 1939, pp. 103-119; also "Mental Deficiency and Crime," in *Social Attitudes*, Young, Kimball (editor), Henry Holt & Company, Inc., 1931, pp. 357-375.

⁵ See Wallin, "Who Is Feeble-Minded"; "Criminal Irresponsibility," *Journal of Delinquency*, November, 1916, 250-255; "Feeble-mindedness and Delinquency," *Mental Hygiene*, October, 1917, 585-590; *Problems of Subnormality*, 1917, particularly pp. 248-250; and "An Investigation of the Sex, Relationship, Marriage, Delinquency, and Truancy of Children Assigned to Special Public School Classes," *Journal of Abnormal and Social Psychology*, April-June, 1922, especially 29 ff.

children have been transferred to the proper kind of special class, the misbehavior has ceased. Reference may be made at this point to one illustrative case among the many hundreds that could be cited.

ILLUSTRATIVE CASE: How a low-grade girl responded when the program was adjusted to her needs in a special class. H.S. was referred to the psychoeducational clinic at the age of 9.9 after five months in the kindergarten and four months in the first grade. She had been excluded because she "had never done any good work in school, and had been disorderly and very hard to control. She was unable to play with the other children, she would hit them and throw stones at them and at passers-by. She would grow angry when her mother reproved her." The examination revealed a Stanford-Binet age of 5.3, an I. Q. of 55, a Sequin form board level of 5.5, severe articulation defects, nasal obstruction, and enlarged postcervical glands. She was subject to headaches and was undergoing blood treatment for venereal infection.

The teacher's report indicates the nature of the transformation wrought by the special class. When she entered she knew nothing about numbers, but after a few months she was able to make figures, she had learned the combinations to 10, and could count by 2's, 5's, and 10's. So interested did she become in numbers that she worked at her arithmetic tasks at home. Her progress in reading had not been so good, but she had improved greatly in oral expression. At first she never spoke in complete sentences. After a year and eight months in the special class she always spoke in whole sentences and articulation had improved notably. She proved to be keen in sense training, was interested in various forms of motor work, was good in various kinds of weaving and knitting, and very good in sewing. Her best work was in sewing and industrial activities. Although limited as to judgment, observation, and general information, she attended well, made good effort, was persistent, retained moderately well, and was fairly dependable. She was neat and clean and, what is more to the point, had never caused any trouble during the entire time she had been in the special class. She had "remained a quiet, courteous, sweet-tempered, helpful, and socially minded little girl who always played nicely with the other children." She had "found herself in the special class, and the special class will make it possible for her to realize her highest possibilities."

The ordinary school work is maladjusted to the capacities, interests, and needs of a large ratio of backward children. It does not adequately prepare them for the most important responsibility confronting them after leaving school, namely, the making of a living by the skillful employment of the powers and capacities which they possess. It does not afford sufficient training, or the right kind of training, for the proper development of these powers. And so we frequently find that many of the dullards in school later gravitate toward the ranks of the social and industrial incompetents, the vagrants, hoboos, paupers, inebriates, malefactors, criminals, and prostitutes. Apparently a much larger proportion of industrial turnover or economic failure is due to mental or educational incompetency and lack of trained skill in craft work than to physical inefficiency or weakness. One industrial establishment discharged 80 per cent of its hired men within four months as "mentally" incompetent. One insurance company which requires a passing grade of 75 per cent for its higher positions, found that 60 per cent of 1,443 applicants failed on the mental tests and only 26 per cent of 1,125 applicants failed on the physical tests. Undoubtedly a considerable part of industrial incompetency is due to the attempt of the schools to force all children into a common mold, instead of supplying appropriate manual and vocational training, especially for those of inferior endowment.

The lack of efficient motor, occupational, and vocational training, however, is probably responsible for more industrial failures than a low intelligence level. The intelligence level of 154 "migrating unemployed" men, many of whom were tramps or hoboos by profession, given the Stanford-Binet by Knollin, varied from seven to eighteen years, the median intelligence age and I. Q. being 14.1 and 89, respectively. The lowest 25 per cent were below 12.6 and 79, and the highest 25 per cent were above 15.6 and 98. Only 5.2 per cent tested below ten years, and only 8.4 per cent below eleven years.⁶

⁶ Terman, Lewis M., and Knollin, Herbert E.

The corresponding figures for Johnson's 107 destitute men, selected at random from those aided by social service organizations, were 5.5 per cent and 12 per cent.⁷ The median intelligence level and I. Q. for both groups, 14.2 and 89, respectively, exceeded the medians for streetcar men, salesgirls, and civil service applicants, but were considerably lower than the medians for business men and railroad engineers.

Definitions

Less effort has been made even now to formulate satisfactory definitions of the backward than of the mentally defective child. Although we may not be able to offer rigidly scientific or exact definitions, the concept of the backward child is entirely justified. There are children who cannot be classified as strictly normal. They are subnormal in varying degrees, but clearly not mentally defective. The same criteria may be suggested for the identification of this large group as were used in defining mental defectiveness, the socio-occupational being considered in the final paragraph.

Psychological definition. Backward children, psychologically, are more or less deficient in all-around mental (*i.e.* intellectual) capacity, ranging for grade children from about a year to three or four years, depending upon the age of the child. In terms of I. Q. Terman places the "borderline" between 70 and 80 (some of these, however, are considered as mentally defective) and the "dull" between 80 and 90. He found that about 10 per cent of his children "of average social status" in various areas in California had I. Q.'s of 85 and less.⁸ The number is somewhat larger for the children tested in different states with the 1937 revision.⁹ Burt, on the basis of an I. Q. of 85, found intellectually backward "rather over 10 per cent of the total school population" in London and Birmingham, at least 20 per cent in the rural

⁷ Johnson, Glenn R., "Unemployment and Feeble-mindedness," *Journal of Delinquency*, March, 1917, 59-73.

⁸ Terman, *The Measurement of Intelligence*, pp. 52, 78.

⁹ Terman and Merrill, *Measuring Intelligence*, p. 37.

areas, and about 15 or 16 per cent of the total school population in Scotland (based on a survey of 100,000 Scottish children).¹⁰ On the same criterion Porteus's ratio was 14.4 per cent for the children in the industrial suburbs of Melbourne, Australia.¹¹

Of 411 consecutive examinees in a clinic for the mentally retarded the author found a median Stanford-Binet I. Q. of 72 for those classified as borderline and 80 for those classified as backward, but the range in each category was very large, as already stated (see Table I).

As has already been emphasized, even a diagnosis restricted to intelligence should not be based purely on I. Q.'s, particularly on I. Q.'s based on verbal tests without reference to performance test results. Diagnoses involving prognoses must always consider the factors of causation, the subject's reaction pattern at the time of the test, particularly his degree of co-operation or lack of motivation, and the degree of improbability as indicated by the synoptic findings.

The groups classified as "retarded" in Table I are assumed to be restorable, whereas those classified as "backward" are, theoretically, improvable but not completely restorable. But this distinction does not always obtain in fact, for some backward cases in the end prove to be restorable, and some retarded fail to reach the normal standard. However, this is merely conceding that errors of diagnosis are not unavoidable.

There are other children who are merely deficient in some specific way—memory, imagination, attention, speed of forming associations, and so on. It seems inadvisable to classify such children as backward if they are normal except for a specific handicap, although the distinction is often difficult to establish.

Educational definition. A child may be specifically or ge-

¹⁰ Burt, Cyril, *The Backward Child*. London: The University of London Press, 1937, pp. 84, 86; *The Subnormal Mind*. London: Oxford University Press, 1935, pp. 117 ff.

¹¹ Porteus, Stanley D., *The Practice of Clinical Psychology*. New York: American Book Company, 1941, p. 404.

nerically and permanently or temporarily (or inherently or accidentally) educationally backward. Burt refers to the English custom of classifying a child as educationally backward who is retarded at least two grades. He classifies as backward all those who in "the middle of their school career are unable to do the work of the class next below that which is normal for their age."¹² Accordingly, a child of ten who cannot do standard III, which is normal for the median nine-year-old (ranging from 8.5 to 9.5) would have an educational ratio below 8.5/10.0 or an E. Q. below 85.

In the bulletin of information recently issued by the British Ministry of Education a child "so retarded that his standard of work is below that achieved by average children 20 per cent younger than he is," is "educationally sub-normal."¹³ On this standard 8 or 9 per cent of primary school children are educationally retarded, exclusive of mental deficient, and a larger ratio of secondary pupils.

F. J. Schonell regards any child as specifically backward and in need of remedial instruction if "his ability in a subject or allied subjects is at least one-and-a-half years below his other educational attainments, and at the same time below his level of general intelligence."¹⁴ On the basis of an investigation of 15,000 English children from average socio-economic districts this standard yielded 5.7 per cent of boys and 2.5 per cent of girls (mean 4.1 per cent) with specific reading disabilities; 5.9 per cent of boys and 2.1 per cent of girls (mean 4.09 per cent) with spelling disabilities; and 5.5 per cent of boys and 4.2 per cent of girls (mean 4.9 per cent) with disabilities in composition (arithmetic was not included in the study).

On the basis of Burt's criterion Lusty estimated that 12.7 per cent of 366 pupils in standards III to IV in New Zealand had a specific educational disability: 8.9 per cent in arith-

¹² Burt, *The Backward Child*, p. 77.

¹³ *Special Education Treatment*. Ministry of Education Pamphlet No. 5. London: His Majesty's Stationery Office, 1946, pp. 19 f.

¹⁴ Quoted from Winterbourn, Ralph, *Educating Backward Children in New Zealand*. New York: Oxford University Press, 1944, p. 76.

metic, 3.4 per cent in reading (half of these in spelling also), and 0.4 per cent in spelling only.¹⁵ In the United States most specific subject matter disabilities are usually reported in reading; ordinarily from 20 to 25 per cent of primary grade pupils experience reading difficulties.

The A. Q. has been suggested as a special device for determining whether a child is educationally backward, or accelerated, in relation to his ability as evidenced by test results (p. 172). Little use has been made of this criterion although it possesses some merit.

RESTRICTED USE OF TERM. We have through the years arbitrarily limited the application of the term educationally backward to children who are all-around backward through inherent lack of ability (as evidenced by test findings and the educational record), and who are in need of instruction, full-time or part-time, in an opportunity class for backward children, as distinguished from a special class for the mentally deficient. The child so defined is unable to make normal progress in school (perhaps only from 35 or 40 per cent to 75 or 85 per cent of normal progress) after an adequate period of legitimate probation: that is, regular attendance, proper classification, skilled instruction, adequate motivation, diligent application, and satisfactory physical health. The educational potentials of these children will vary from about third or fourth grade to fifth or sixth grade, or even higher, depending on the length of time they continue in school.

DIFFERENTIAL DIAGNOSIS. This definition is intended to exclude (a) children who possess fair or good ability or intelligence, but who are specifically deficient in only one or two branches (or in the mental traits on which success in such branches depends)—for example, reading or arithmetic—and who are in need of remedial instruction in their weak branches. These children might be assigned to special sections or remedial groups in those branches, but do not require full-time assignment to an opportunity class. Most children

¹⁵ *Ibid.*, p. 77.

are not equally capable in all arts, sciences, and handicrafts; indeed many persons, even those conspicuously well endowed in general, are often deficient in some type of subject. (b) Children who are educationally retarded merely because of unfavorable extrinsic factors—lack of cultural environment, neglect, abuse, absence from school, frequent transfers, lack of interest, application, and willingness to learn, and inferior teaching. Such children may be brought up to grade if they are given adequate skilled instruction in time. If neglected too long, their potentials will probably remain undeveloped or will stagnate or undergo “functional atrophy” so that they will, to all intents, be like the child who is genuinely deficient in inherent capacity.

Mental defect from social deprivation. The conditions under which social isolation will produce mental defectiveness—that is, the duration and severity of the neglect and the time at which it occurs in the child's life—are not known. Davis refers to the case of Edith Riley, who, at eight years of age, had been incarcerated for some years in a closet. She lost her capacity for speech and vision but recovered “complete normality within two years.”¹⁶ A more recent case of extreme social deprivation chronicled in the press in this country is that of Anna, whose spindly, emaciated body was discovered at about the age of five wedged in an old broken chair tilted backward in a dirty second-floor bedroom without sunshine in a farm house in Pennsylvania. The second illegitimate child of a subnormal unmarried mother, she had been isolated from the rest of the mother's family on the demand of the outraged grandfather. No one ever spoke to her. She had not been bathed and had merely been fed milk until she became five years of age, when she was given some oatmeal. Unable to talk, or walk, or even move, she was placed successively in a county home, a private home, and a small school for mental defectives. As a result of proper

¹⁶ Davis, Kingsley, “Extreme Social Isolation of a Child,” *American Journal of Sociology*, January, 1940, 554-565.

physical and educational care she became chubby (in fact, 20 pounds overweight), learned to walk, could feed herself, responded to verbal commands, and sought attention, but she was unable to talk at the age of seven and a half. Davis interprets this as a case of idiocy through social deprivation, and believes that earlier treatment would have restored her to mental normality. She was reported by observers to have been a normal, even beautiful baby. He apparently believes that five years of physical and social neglect in early life is sufficient to make it "impossible for any child to learn to speak, think, and act like a normal person."¹⁷

Eugenic definition. There is a type of backwardness which is due to cacogenic heredity and is transmissible just as are some kinds of feeble-mindedness, insanity, epilepsy, or any other heritable neuropathic condition. The laws of heredity, whatever they are, are the same for all types and grades of human beings. Acquired forms of backwardness that are due to various kinds of destructive environmental forces are not transmitted unless the germ plasm has been modified.

That one's characteristics are the joint product of the interaction of the genes and the intrauterine and the external environment is now admitted. Whether the genes can be mutated (modified) by so-called blastophthoric substances and whether the mutations thus produced can be transmitted to subsequent generations continues to be debatable. Experiments (p. 214) have shown that it is possible to produce transmissible gene mutations in plants and in some animals by means of X-rays and other short-wave radiations and that chromosome changes, such as doubling, deficiencies, or translocations, can be produced in flies from the use of the alkaloid poison, colchicine. Although it has not been definitely demonstrated experimentally that gene mutations in human beings can be produced by the ingestion of alcohol (often called a racial poison) and lead, it is pertinent to suggest that the

¹⁷ *Ibid.*, p. 564.

possibility remains that many generations of human alcoholization might produce cumulative changes tending to impair the germinal cells.

Anatomical definition. Anatomically there are two distinguishable types of backwardness: one due to hampering physical defects, such as defective nutrition, obstructed breathing, auto-intoxication, diseased caries; and the other due to a certain amount of deficiency in the cellular tissue of the brain. It is probable that the cerebral neurones are not so sensitive or responsive or so finely organized in the brains of most children who are genuinely backward.

Differences between the mentally defective and the backward

What then, in brief, are the essential differences between mentally defective and backward children?

(1) From the point of view of the character of the mental defect, the difference is primarily quantitative. The mental defective possesses the same outline of mental functions, but the functions are less highly developed or organized, or they are more seriously impaired or crippled. The primary group of mental defectives simply form the fag end of the curve of distribution. The qualitative differences, particularly apparent in the special clinical types, can, perhaps, be regarded as expressions of extreme quantitative departures or as more involved patterns of traits.

(2) From the point of view of curability, genuine mental defectiveness of the hereditary type is ineradicable, according to our present knowledge, with possibly insignificant exceptions. Most of the exceptions pertain to certain pathogenic cases in which the early arrest of a disease process might prevent mental defect. Constitutional mental defectiveness can only be ameliorated by appropriate educational and social training and hygienic care. In the case of backwardness, the outcome depends entirely upon the type of deficiency. If grave deficiency or imperfection exists in the cellular organi-

zation of the brain, the defect probably can never be wholly effaced or overcome. There will probably be a gulf between the mind of such a backward child and that of a normal child that never can be wholly bridged by hygiene, medication, or education. And yet, these children can be improved far more than the mentally defective by systematic exercise of the sensori-motor and intellectual functions, by developmental and remedial literary instruction and craft training, and by proper hygienic care. If the backwardness is due to educational and social neglect, it can sometimes be entirely overcome by appropriate instruction and training given in time. Children who are backward because of ill-health, disease, hampering physical defects, and abuses (indulgence in alcohol, tobacco, and other injurious practices) may sometimes be restored to complete normality by the improvement of the physical condition through the application of the proper remedies and corrective treatment. Some writers, however, believe that the orthophrenic (mentally ameliorative) effect of the removal of physical defects has been exaggerated.

Orthophrenic effects of physical therapy. The evidence and the claims are conflicting. Some physicians are inclined to attribute all mental retardation to physical defects and to promise mental restoration from the removal of adenoids and tonsils. Some years ago a press release in a New York daily stated that an experiment in Pontiac, Michigan, had shown that "infected tonsils are an important cause of moronism." Seven hundred and thirty-six children who had had diseased tonsils removed were compared with 741 unoperated children with infected tonsils who served as controls. Follow-up studies at intervals of from six months to two years revealed that between 91 and 94 per cent of the treated group showed marked mental and physical improvement, whereas "no similar turn for the better" was found among the controls. Only 57 of 148 cases of "subnormal mentalities and backwardness in school work failed to show improvement, while the rest showed remarkable benefits." Unfortunately, the seat of the

mental or educational difficulty frequently is higher up than the nasopharynx!

In one of the earliest studies of the effects of the removal of diseased tonsils in school children, MacPhail found that only one-third evidenced improvement in their school work during the ten-month school period following the operation as compared with the ten preceding school months, whereas slightly over half actually did poorer work. On the basis of all the work done, although a decline was apparent immediately after the operation, a marked improvement was eventually discernible which was not paralleled by an unselected group of children whose percentage of improvements remained stationary from year to year.¹⁸

Two years later two studies appeared based on different procedures with varying results. Rogers measured the height, weight, dynamometry, speed of tapping, and the intelligence (I. Q.) of a test group of boys who had had their adenoids and tonsils removed and an unoperated control group of corresponding intelligence and nasopharyngeal condition. The terminal measurements, given about a year after the operation, showed that the relative improvement of the operated cases was greater only in weight and speed of tapping. Since the increase in the I. Q. was not greater for the operated than for the nonoperated cases, and since the distribution of I. Q.'s was the same for a larger group of tonsil cases as for a group of non-tonsil cases, she concluded that adenoids and enlarged tonsils do not cause mental retardation, although granting, apparently, that they may affect the child's educational efficiency.¹⁹

Mallory's results were more positive. He measured the degree or coefficient of association (based on Pearson's association formulae) between five major physical defects (in-

¹⁸ MacPhail, A. H., "Adenoids and Tonsils. A Study Showing How the Removal of Enlarged or Diseased Tonsils Affects a Child's School Work," *Pedagogical Seminary*, June, 1920, 188-194.

¹⁹ Rogers, Margaret C., "Adenoids and Diseased Tonsils: Their Effect on General Intelligence," *Archives of Psychology*, No. 50, 1922.

volved the tonsils, nose, teeth, eyes, and ears) and achievement scores in standardized educational tests in the case of 515 children, distributed through the elementary grades. The physical examinations were made by a registered nurse. The children were classified into four groups: physical defectives with low scores and with high scores in the achievement tests, and sound pupils (nonphysical defectives) with low and high scores in the achievement tests. Intelligence was measured by the Illinois Intelligence and the Holley picture tests. After freeing the results by partial correlations from the influence of intelligence, school attendance, and retardation (as determined by the educational quotient), the coefficients of correlation revealed a direct relation between physical defects and low achievement scores, the defects ranking in the following order: nasal obstruction, decayed dentures, defective hearing, defective tonsils, and defective eyes. The influence of eye defects and defective vision was almost negligible.²⁰

Six years later Hoefer and Hardy made a three-year study of the psychological and educational improvement of a random sample, economically and socially considered, of 343 white pupils, ages eight to eleven, from grades 3B, 3A, and 4B, with I. Q.'s above 74, who had been classified by the same pediatricians into four groups on the basis of their general physical condition. Note was made of the condition of the adenoids and tonsils, and data were also taken on tonsillectomies and the practice of coffee drinking. Conclusions regarding psychological and educational growth and improvement were based on the results of the Binet-Simon and the Primary and Advanced Form A of the Stanford Achievement Test. Although the differences did not meet the "statistical standard for reliability," the I. Q. gains were rather consistently better for the better physical group, 4.79 points com-

²⁰ Mallory, Jasper N., *Study of the Relations of Some Physical Defects to Achievement in the Elementary School*, Nashville, Tenn.: George Peabody College Contributions to Education, No. 9, 1922.

pared with 1.78 for the poorest group. In the educational tests no consistent gains were apparent except in spelling. Nevertheless, those in the best physical condition had the "highest ratings in intellectual and educational achievement." Those with tonsillectomies before the investigation started ranked highest—and maintained their superiority—and those with diseased conditions the "lowest in every instance." The "non-coffee group ranked highest in every instance." They showed "definite superiority" over coffee drinkers and over those who reported an increased amount of drinking.²¹

This study was followed in 1931 by Laird, Levitan, and Wilson's investigation of 53 children, selected by the teachers as the most nervous in their classes, from the first, third, and fifth grades of three schools, although they were free from malnutrition or other serious defects. The extent of nervousness was measured by Willard C. Olson's Behavior Check List. The investigation disclosed that the nervous behavior symptoms could be overcome by removing hunger pangs by means of a daily school lunch. The children were divided into three groups: a control group which did not receive any feeding; an experimental group given a pint of milk at 9:30 a.m.; and a second experimental group given a half pint of milk plus a calcium metabolism concentrate (calcium, maltose, lactose, phosphorus, vitamin D, and some cocoa) at the same hour. At the end of a two weeks' period, the improvement amounted to 2.3 per cent for the control squad, 8.2 per cent for the first experimental group, and 15.6 per cent for the group given the concentrate. Eighty-five per cent of the latter group manifested marked improvement as measured by the Check List—25 per cent or more for 15 traits.²²

The problem of prenatal maternal malnutrition and post-

²¹ Hoefer, Carolyn, and Hardy, Mattie C., "The Influence of Improvement in Physical Condition on Intelligence and Educational Achievement," *Twenty-Seventh Yearbook*, Part I, 1928, pp. 371-387.

²² Laird, D. A., Levitan, M., and Wilson, V. A., "Nervousness in School Children as Related to Hunger and Diet," *Medical Journal and Record*, 1931, 134: 494-499.

natal malnutrition and the improvement of behavior reactions through improved nutrition and the administration of vitamin B₁ (Thiamine), vitamin B₂ (riboflavin), benzedrine, and glutamic acid are reviewed in Chapter 8.

The relation of deficient or abnormal mentation to endocrinopathic disorders and improvement from glandular therapy is considered especially in Chapters 10 and 12. The effectiveness of glandular treatment varies with the age of onset of the disorder, the type of disorder, the degree of the dysfunction, and the timeliness of the treatment. The slighter degrees of endocrine disturbances are probably more common and more responsive to medication than the graver disorders. Gordon, Kuskin, and Avin report an improvement in 45 per cent of 155 endocrine cases among 317 mentally retarded children who received organotherapy coupled with betterment of associated disorders, educational measures, proper diet, and the improvement of social conditions. The exact degree of improvement from the administration of glandular extracts is indeterminate. However, improvement in the case of 162 nonendocrine children was only 1.2 per cent.²³

A quarter century earlier an attempt was made by the author to measure by means of a pioneer set of group psychological tests the amount of improvement made by a group of children receiving dental treatment to meet the conditions found in the oral cavity. A squad of 27 school children suffering from diseased dentures and gums, an insanitary oral cavity, and frequently from defective nutrition, was given appropriate dental treatment, including fillings, extractions, treatment of the gums, demonstrations in brushing the teeth and gums, and instruction on keeping the mouth sanitary and in chewing food. Two demonstration meals were arranged to show correct eating habits. The results of the treatment were

²³ Gordon, M. B., Kuskin, L., and Avin, J., "Organotherapy in Mental Retardation Associated with Endocrine and Non-Endocrine Conditions," *Endocrinology*, 1935, 19: 572-578. Many instances of mental improvement from glandular therapy are given in Mateer, Florence, *Glands and Efficient Behavior*, New York: D. Appleton-Century Company, Inc., 1935.

Chapter 6

CLASSIFICATION ACCORDING TO PSYCHOLOGICAL CHARACTERISTICS

Diagnosis and classification

The first step in a scientific program of rehabilitation of children who are handicapped in any way is to obtain a correct understanding or diagnosis of the defect or the handicap and of the personality make-up and reaction characteristics of the deviate. A diagnosis (*dia*, through or apart; *gnosis*, knowledge) refers to any examination procedure by means of which a condition or disorder, whether physical, psychological, educational, or social, can be thoroughly understood (known through and through), and correctly distinguished from other disorders more or less closely related. A scientific classification is the essential starting point in any scientific system of remedial work, because an adequate and correct classification presupposes a correct diagnosis. The moment you have accurately classified your deviate you have, at the same time, diagnosed his condition, provided the classification is based, first, on a correct determination of the nature of the difficulties found by systematic, clinical observations (symptomatic diagnosis) and by means of laboratory tests or standardized objective test procedures (diagnosis by controlled diagnostic tests), and, second, on the discovery of their causes (causal or etiological diagnosis). No classification of human deviations is complete or satisfactory that is not based upon the discovery of the underlying causative conditions or

factors. The classification and treatment of diseases remains purely empirical and symptomatic until the causative factors have been uncovered and removed. Diagnosing a pain in the head as a headache and abolishing the pain by means of some anodyne or analgesic, like aspirin, is purely empirical and is merely treating symptoms instead of finding and removing causes. One physician has listed over a hundred causes of headache, some of which require radically different treatments. Reducing a fever by means of a febrifuge is a relatively simple procedure that requires little diagnostic skill. Such treatment is purely symptomatic and may possess only dubious value until the specific causal treatment is applied, perhaps to a pathological condition in the throat (diphtheria) or in the intestinal membrane (typhoid fever). Similarly, the suppression of the tendency to pilfer or fib by punishment, or the overcoming of a reading disability by added drill on ordinary instructional procedures is symptomatic rather than causal treatment. Although symptomatic treatment sometimes proves effective in removing symptoms, at least temporarily, it may not reach the root of the trouble and effect the radical reconstruction needed for a permanent cure.

Not only should the diagnosis reveal the nature of the disorder and its causation, but it should also indicate the line of differential treatment needed to correct the disability, whether medical, surgical, educational, psychological, or social. A diagnosis of pulmonary tuberculosis indicates one kind of medical and hygienic treatment; a diagnosis of typhoid fever indicates another form of therapy; a diagnosis of diphtheria points to a quite different form of medication. Similarly a psychological classification of imbecility indicates the kind of educational training, social care, and guidance that a child of imbecile level requires. In the case of speech defects, if the diagnosis is a sibilant lisp, the kind of corrective articulation training needed is known. A classification of stuttering or stammering suggests a markedly different thera-

peutic approach. Different kinds of reading disabilities or difficulties require the application of different kinds of remedial reading techniques.

Obviously the matter of a correct classification or diagnosis is of prime importance, not only in the field of medicine, but also in the field of education. To the public school educator it indicates whether the child should be assigned to some kind of special class—for the mentally deficient, the backward, the partially sighted, the deaf, or the crippled—or be afforded curriculum modification to meet his level of ability or special interests; or be provided with some form of therapy to overcome a specific educational disability; or should receive discriminating psychological or psychotherapeutic treatment to correct some form of personality maladjustment or emotional handicap. In the case of a mental defective transferred to a colony, a correct classification indicates whether the inmate should be assigned to the hospital for medical treatment, or to the school department for pre-academic, academic, or occupational training, or to the occupational division for cottage, shop, or farm employment. The importance of providing an examination of the brain condition of alleged mental defectives by modern brain examination techniques before any steps are taken to send such children to special classes or state colonies is emphasized by Abraham Levinson, chief of the division of child neurology and psychiatry in the Cook County Hospital, in the following words:

Encephalography [an X-ray technique for the examination of the brain in living persons, explained in Chapter 8] is helpful in the making of a decision as to the disposition of the case: whether the child is to be institutionalized, sent to a special school, or be kept at home. It is a mistake to commit a child to an institution on the supposition that he is uneducable. I do not recommend that a child be sent to a state institution for mentally defectives [*sic*] without first doing an encephalography on him. The fact that most of our state institutions are greatly undermanned and that they have no adequate provision for the care and instruction of borderline cases is reason enough for making certain just what

is the status of the child's brain, before committing him to an institution. . . . Parents of mentally deficient children are almost without exception deeply sensitive about the mental status of their children. They often look upon the retarded condition as a reflection upon themselves. They like to delude themselves into thinking that things will improve and find it difficult to face the truth. No amount of argument is effective. However, when they are confronted with the encephalography plate that shows them definitely that there is pathology that is not remediable, it is more effective than any lengthy argument. A nontechnical explanation of the encephalogram is time consuming, but very much worth while.¹

It is tragically true that thousands of children in years past have been transferred to special classes or committed to residential institutions without adequate examinations by competent diagnosticians. A misdiagnosis of mental defectiveness is often attended by most unfortunate consequences for the child or for the parents, because it may cause doubts, anxiety, heartaches, resentment, or self-incrimination in the parents, or it may stigmatize the child, at least in the estimation of sensitive parents, or it may lead to lifelong incarceration in a state colony or private residential institution. The matter of a correct diagnosis and classification must, obviously, be given a position of primacy in the program of adjustment for deviating children. The more accurate the diagnosis, the more intelligent and effective will the treatment be for all kinds of handicapped children without exception.

Logically, therefore, our first concern when a child enters school is to obtain reliable information regarding his psychosomatic condition and needs from comprehensive medical and psychological examinations. The data from thorough physical and mental examinations should make it possible to give early attention to his physical handicaps, so that his physical needs may receive prompt attention, and to plan an

¹ Levinson, Abraham, "Pneumoencephalography in Mentally Deficient Children," *American Journal of Mental Deficiency*, July, 1947, 1-8 (contains pneumoencephalograms of different kinds of mentally deficient children).

educational program from the very outset more nearly in conformity with his peculiar physical, mental, educational, and social requirements. If the child could be educationally classified (or diagnosed) with absolute accuracy at the time of enrollment, his educational treatment could be planned accurately from the start of his school career. Of course, this cannot be done with a high degree of accuracy in our present state of knowledge, partly because the problems of child growth and development are highly complicated and abstruse, partly because the causation of mental defects, deviations, and personality maladjustments often is very complex and obscure, and partly because our psychological and educational (as well as medical) examination techniques are far from perfect. This must be frankly admitted, in spite of the great progress that has been achieved since the first decade of the century in the construction of numerous kinds of standardized objective tests and measuring scales. These tests and scales, however crude they may be, enable the trained examiner to measure objectively with a fair degree of accuracy the amount of many psychic, psychophysical, and educational traits that children possess, and to differentiate various kinds of deviations and many levels of capacity and achievement. Unquestionably the numerous batteries of psychological and educational tests now available, in spite of their imperfections, enable educators to obtain a more comprehensive and accurate picture of the child's mental and educational condition at the time of observation and to classify him for purposes of educational adjustment more accurately than was possible with the procedures which were previously used. These nontechnical procedures include unaided observation of the child's behavior patterns, his facial appearance, and other bodily characteristics (especially size); the notation by the medical inspector of outward neurological (nervous) signs and symptoms and physical defects and diseases; the discovery of hereditary defects in the ancestry by means of pedigree or genealogical investigations; and the investigation of his school

history, especially his record of educational retardation or grade repetition, failures in various kinds of subject matter, academic grades, and the results of standardized group tests of capacity and achievement. But a physical examination, however important from the standpoint of disclosing diseases or defects that may be related to the child's mental, educational, or social maladjustments and may yield to medical or surgical treatment, does not supply a reliable measure in itself of the kind and amount of the child's mental deviations or of his personality idiosyncrasies. Personal and family histories, however important in certain cases, frequently are too barren, obscure, or involved to yield unambiguous clues regarding the genesis and nature of the child's handicaps. Nevertheless, comprehensive case study procedures based on physical, psychological, psychiatric, educational, genetic, and social investigations have brought cosmos out of chaos, relatively speaking, in our erstwhile confused and discrepant psychological and educational classifications, and have made it possible to differentiate many kinds of educational deviates with a fair degree of accuracy and to prescribe the kinds of differential educational therapy appropriate for each kind of handicap.

The multiphasic nature of a satisfactory classification of mental deviations. A measurably complete classification of children with mental and physical handicaps, particularly mentally deficient and retarded children, should include information from every important field of inquiry. At least five aspects or points of view are of primary importance in the classification of mental subnormals: (1) the nature of the cause (referred to as etiology); (2) special physical or clinical types (which are very numerous); (3) psychological characteristics or criteria; (4) degree of educability, specific educational abilities and disabilities, and educational characteristics; and (5) socio-occupational competency. Although adequate definitions and descriptions of mental deficiency and backwardness will contain elements from all of these

phases of the problem, all five are not of equal importance in the formulation of the concepts. The five classifications are correlative and supplementary, not mutually exclusive. Every aspect contributes unique data to a comprehensive understanding of the problem of mental deviation from normality.

Criteria employed in psychological classifications. How may mental deficient be classified psychologically? In the past, classifications have been based on a great variety of traits or functions. The following represent, briefly, the most important of such classifications. The classifications based on single traits or functions will be considered first.

According to contrasting emotional or temperamental characteristics

The classical grouping places all the mentally defective under two headings: (1) the unstable, mobile, restless, excitable, hyperkinetic, hair-trigger, psychopathic, disciplinary type; and (2) the apathetic, placid, slow, inert, asthenic type. The unstables are sure to be referred to the psychoeducational or mental hygiene clinics for examination and transfer from the class, whereas the quiet, apathetic cases are often permitted to remain, perhaps in the corner of the room, as chairwarmers. This dichotomous classification, however, is too rigid. Some defectives cannot be classified in either group. They are rather neutral or indifferent, being neither very unstable nor very phlegmatic. Some belong in a mixed group, being sometimes excitable and sometimes dull and listless. Many defectives are like many normals; they have their ups and downs, their episodes of sluggishness and impulsiveness. These contrasting emotional characteristics exist, in varying degree, in all kinds of individuals, normal and abnormal; but they are particularly characteristic of the neurotic, psychotic (mentally disordered), and mentally defective.

The so-called psychopath. During the present century voluminous descriptive accounts have appeared of a type of

child whose chief defect is emotional, "affective," temperamental, or volitional rather than intellectual. He is inclined to be restless, irritable, excitable, impulsive, deficient in volitional control or power of inhibition, lacking in the feelings of gratitude, pity, affection, sympathy, or social solidarity. He is selfish, morbidly egotistical, suspicious, frequently morally insensible, callous, incapable of feeling shame or remorse, viciously or criminally inclined, crafty, scheming, frequently pathologically mendacious (lying without motive or intelligible end), thieving, wantonly destructive or cruel, sexually precocious or abnormal, sometimes sadistic (receiving sexual enjoyment from inflicting severe pain upon the victim), and he is usually more or less deficient in intelligence, but may be normal or supernormal.

The above characterization may seem to be overloaded with descriptive adjectives, but it does scant justice to the profusion of terms² that have been applied to a type of defect which some believe constitutes the very core of character or personality defect, and the central defect in a type of individual regarded as the potential criminal par excellence and constituting the largest single group of the actual criminal class. These individuals are looked on as abnormal if not pathological, well-nigh incurably antisocial, and partly or wholly irresponsible.

Various terms have at different times been applied to this type of personality defect: constitutional immorality (Tanzig); moral insanity (Müller, Mendel, Tiling); moral idiocy (Brunet); moral imbecility (Grohmann); psychopathy, or psychopathic or constitutional inferiority. An English stat-

² With the use of the Rorschach test other characterizations are coming into vogue. Thus, Bowlus and Shotwell, on the basis of a Rorschach study of a dozen girls who had been institutionalized as "defective or psychopathic delinquents," concluded that the outstanding traits of the psychopath are: frivolousness, coyness, flightiness, sketchiness, and self-centeredness. These terms are substituted for Robert M. Lindner's earlier "superficiality, avoidance, explosiveness, incompleteness, and egocentricity." Bowlus, D. E., and Shotwell, Anna M., "A Rorschach Study of Psychopathic Delinquency," *American Journal of Mental Deficiency*, July, 1947, 23-30.

ute, for many years, until 1927, gave legal sanction to the concept of moral imbecility. Moral imbeciles were defined as "persons who from early age display some permanent mental defect coupled with strong vicious or criminal propensities on which punishment has little or no deterrent effect." The underlying assumption was that these children, especially when the vicious tendencies appeared early in life (and some would not employ the category unless the tendencies did appear early), are congenitally defective in the impulses, feelings, and sentiments which are at the base of the so-called moral sense, and that therefore the antisocial tendencies are largely ineradicable. Some believed that these children suffered from a specific moral or ethical defect: they were "morally color-blind," and the defect showed itself in vicious, incorrigible conduct. The delinquent children were treated as defective in a sense.

Criticism. It may be doubted whether there are any moral imbeciles in this sense. In the writer's block of 2,774 consecutive clinic cases only one was diagnosed as a "moral imbecile" (though more had been so reported), but his conduct so improved in the course of a few years as to render the diagnosis questionable. Among the mentally defective, only 1 per cent are moral imbeciles, according to Mary Dendy; only 5 per cent are "vicious, immoral and antisocial," according to A. Eichholz; and less than 4 per cent of the mentally defective in "lunatic asylums" are morally defective, according to the 1909 report of the Commissioners in Lunacy in England. James Kerr considers that the prevalence of moral imbecility is very small and W. C. Sullivan, that only a "very small amount" of juvenile criminality can be attributed to it. Mönksmöller, however, classified as moral imbeciles 13 of 200 young offenders examined in the Lichtenberg reformatory. Most of these cases probably were mentally defective, insane, hysterical, or psychopathic. The British diagnosticians apparently have made little use of the term. In the 1927 revision of the Mental Deficiency Act the term has been discarded entirely and the term "moral defectives" has been

substituted for it. Moral defectives are defined as "persons in whose case there exists mental defectiveness coupled with strongly vicious or criminal propensities and who require care, supervision, and control for the protection of others." Nothing is said about punishment having "little or no deterrent effect." Although the revised definition marks an improvement, its restriction to cases of "mental defectiveness" limits its usefulness, because many intractable behavior cases are not mentally defective in the traditional sense of that term, but may be intellectually normal or bright.

In the United States, where the terms moral imbecility, moral idiocy, moral insanity, or constitutional immorality never attained much currency, a strong tendency developed in the second decade of this century to classify under such categories as psychopathy (less frequently neuropathy), psychopathic personality, or constitutional inferiority all individuals who display emotional, instinctive, temperamental, or volitional defects. This is especially the case when they evince criminal propensities, and when they cannot be classified as outspokenly psychotic (insane), or psychoneurotic (neurasthenic, hysterical, psychasthenic, migrainous), or epileptic, or mentally defective (though some are classed as both mentally defective and psychopathic). Although the terms psychopath and psychopathy still occur very frequently in the writings on psychopathology and delinquency, it has not yet been clearly established that there is a definite nosological (nosology, classification of diseases) or diseased entity corresponding to these terms, or whether they are merely convenient descriptive terms for a group of ill-defined symptoms not classifiable under any of the well recognized categories. Thus Alexander, psychoanalyst, in his description of the psychopath, concedes that there is "no clear psychiatric diagnosis" of psychopathy; it must be diagnosed "intuitively, through psychiatrically trained insight"; it does not fit into any "definite group of neuroses or psychoses."³ The in-

³ Alexander, Franz, "Mental Hygiene and Criminology," *Mental Hygiene*, October, 1930, 869 f.

evitable result of diagnoses based on subjective impressions is confusion and disagreement. "Psychopathic personality," say Bowlus and Shotwell, "is the diagnostician's wastebasket," for although the psychopaths are "neither sane nor sensible nor intelligent," neither are they "psychotic nor psychoneurotic nor feeble-minded."⁴

In the ultimate analysis, stripped of all unessentials, psychopathy represents to psychologist Cason merely a "serious lack of the ability to control primitive drives and antisocial modes of behavior,"⁵ which may affect all levels of intelligence. It is not a psychosis, a neurosis, or merely an emotional disorder, nor is it necessarily permanent or hereditary. The individual is a psychopath if he has the symptom of "lack of adequate control" of primitive, antisocial drives, regardless of the etiology or effects of the treatment, or of how long or short a time the condition continues. Obviously the category is purely descriptive.

According to psychiatrist Karpman, psychopathy is a "myth and is a nonexistent entity."⁶ The concept has been envisaged purely on the descriptive level and not on the "dynamics back of the symptoms." The cases included in the category "have nothing in common." Nearly all of the so-called psychopaths fall within the framework of some other cardinal clinical group. Psychopaths are either psychotics (often representing the incubation stage of dementia praecox or manic-depressive psychosis) or neurotics with antisocial tendencies. Many are "neurotics with at best only a psychopathic facade." A small group with "primary or idiopathic psychopathy" (which Karpman calls anethopathy) remains, in whom no motivations or psychogenetic factors can be discovered, as can be done in the case of neurotics and psychotics

⁴ Bowlus and Shotwell, "A Rorschach Study of Psychopathic Delinquency," pp. 23, 25.

⁵ Cason, Hulsey, "The Concept of the Psychopath," *American Journal of Orthopsychiatry*, April, 1948, 297-308.

⁶ Karpman, Ben, "The Myth of the Psychopathic Personality," *American Journal of Psychiatry*, March, 1948, 523-534.

who are, therefore, amenable to psychotherapeutic treatment. They therefore "are as close to the constitutional as can be found." These few cases of true psychopathy evidence a "special personality organization having in particular a virtual absence of any redeeming social reaction: conscience, guilt, binding and generous emotions, etc., while purely egoistic, uninhibited instinctive trends are predominant." They are "all antisocial with no redeeming positive social traits." They are not "reteachable or amenable to deep psychotherapeutic approach," but if put in a controlled situation "can be handled with greater ease."

Karpman's trenchant criticisms bring vividly to mind the author's warnings against the indiscriminate use of the vague concept of psychopathy, issued about a third of a century ago, based on research in writings and clinical experience with thousands of mental defectives, psychotics, neurotics, unstabiles, and delinquent children of all kinds. The warnings were about as futile as those against the loose use of the concept of moronity, discussed elsewhere. For example:

IN 1916: [The concept of] psychopathic constitution is more subjective than the concept of feeble-mindedness. . . . Our fear is if we overwork the concepts of feeble-mindedness and psychopathic constitution (as well as constitutional inferiority) and irresponsibility, we shall lay the foundation for eventual reaction which will destroy the general progress which has been made.⁷

IN 1920: The term psychopathic constitution—with all its equivalent expressions—has not yet been precisely enough defined to be used without ambiguity in diagnosis. Many of the leading psychiatrists do not agree as to what constitutes a psychopathic constitution. The numerous signs and symptoms that are thus diagnosed have by no means the unmistakable character of a positive Klebs-Loeffler or a positive Wassermann. This is undoubtedly the principal reason why some medical examiners report a large percentage of "psychopaths" among delinquents, while others never find a case. The large differences found are probably subjective rather than objective. . . . Until this field

⁷ Wallin, "Criminal Irresponsibility," *Journal of Delinquency*, November, 1916, 254, 255; also *Problems of Subnormality*, p. 119.

has been more fully investigated, the examiner who desires to be cautious must employ the concept of psychopathy guardedly.⁸

IN 1922: The inexorable logic of facts has brought a sharper limitation of the concept of feeble-mindedness than the most conservative of us conceived possible a few years ago. It will probably be possible only through a similar inescapable logic of inexorable facts to set a reasonable limitation to the category of *psychopathy*, which has attained sudden popularity as a result of the limitation of the feeble-minded group. Since no one has been able to supply a scientifically adequate pathognomy of a nosological entity corresponding to a psychopath, would it not be prudent merely to refer for practical purposes to the emotionally unstable or ill-balanced and volitionally weak delinquent who is neither feeble-minded nor insane, as a "defective delinquent," without making any assumption regarding a "diseased entity" called psychopathy, which may prove to be mythical? ⁹

IN 1923: In spite of the voluminous dialectics published on the psychopath, I have been forced to assume an attitude of caution in the practical use of the category. Many of the subjects who have come to my clinics diagnosed as psychopaths have been, according to the teachers' reports, indistinguishable from the general run of cases in the different special classes. Their record of conduct disorders was no worse. Many who had previously been obstreperous, unstable, temperamental, emotional, "psychopathic," rapidly settled down in the special schools. We have not found the pathognomy of psychopathy based on emotional, instinctive, conative, and conduct instabilities sufficiently clear-cut to insure uniformity of diagnosis among different examiners. Nor have we found that the symptomatology of scattering or intellectual irregularity or variability possesses very high pathognomonic importance.¹⁰

With this welter of discordant views regarding the interpretation of the term, it is inevitable that the ratio of criminals and delinquents who have been classified as psychopathic

⁸ Wallin, "The Problems Confronting a Psycho-Educational Clinic in a Large Municipality," *Mental Hygiene*, January, 1920, 103 ff.

⁹ Wallin, "An Investigation of the Sex, Relationship, Marriage, Delinquency and Truancy of Children Assigned to Special Public School Classes," *Journal of Abnormal Psychology and Social Psychology*, April-June, 1922, 34.

¹⁰ Wallin, "The Diagnostic Findings from Seven Years of Examining in the Same School Clinic," *Journal of Delinquency*, May-July, 1923, 190.

will show marked diversities. The following estimates illustrate the confusion or difference of opinion that has existed among psychiatric examiners.

TYPE OF CASE	YEAR	AUTHORITY	PER CENT PSYCHOPATHIC
350 adult criminals (Boston Municipal Courts)	1915	Anderson ^a	24.3
400 women criminals (Mass. Reformatory)	1915	Spaulding ^b	7.7*
608 men prisoners (Sing Sing)	1918	Glueck ^c	18.9
Women criminals (Ill. Reformatory)	1919-29	Sutherland ^d	88.3
Offenders in Detroit (Recorders Court)	1924	Raphael ^e	36.
2,000 juvenile delinquents (Boston, Judge Baker Foundation)	1927	Healy and Bronner ^f	2.8
1,000 juvenile delinquents (follow-up of Judge Baker Foundation cases)	1934	Sheldon and Eleanor Glueck ^g	1.9†
Federal penitentiary criminals (Lewisburg, Penna.)	1937	Wholey ^h	14.
9,958 prisoners convicted in the Court of General Sessions, New York City		Bromberg and Thompson ⁱ	6.9

* Marked neuropathic or psychopathic cases.

† Exclusive of "constitutional inferiors."

^a Anderson, V. V., "The Laboratory in the Study and Treatment of Crime," *Journal of Criminal Law and Criminology*, March, 1915, 840 f.

^b Spaulding, Edith R., "The Results of Mental and Physical Examinations of Four Hundred Women Offenders," *Journal of Criminal Law and Criminology*, January, 1915, 704 f.

^c Glueck, Bernard, "A Study of 608 Admissions to Sing Sing Prison," *Mental Hygiene*, January, 1918.

^d Quoted from Sutherland, E. H., *Principles of Criminology*, 1939, p. 110.

^e Raphael, T., *et al.*, "Socio-Psychiatric Delinquency Studies from the Psychopathic Clinic of the Detroit Recorders Court," *American Journal of Psychiatry*, April, 1924.

Among 2,774 school children examined in the psychoeducational clinic in St. Louis who were diagnosed by the writer on the basis of the synoptic findings, only 1.18 per cent were classified as psychopathic.¹¹ The original data are not now available for the group of children who were subject to some form of conduct disorder (20 per cent of the entire number), but the proportion classified as psychopaths was not much higher. A much larger ratio, 6.7 per cent, of all these examinees were classified as nervous and neurotic. That many emotionally unstable and delinquently or criminally inclined children and adults exist is incontrovertible, but it adds little to our understanding of the dynamics or of the handling of the problem of misbehavior or criminality in the intellectually normal or subnormal to apply the label of psychopathic.

According to specific or general motor capacity

Hack Tuke classified the mentally defective into (1) those capable of reflex movements only, (2) those capable of ideomotor and emotional responses, and (3) those capable of volitional acts. Such a classification, though suggestive, is not sufficiently precise to be of much practical service for determining the efficiency of the motor apparatus or for properly grading and differentiating psychomotor capacities and peculiarities of subnormals. Psychologists have devised and standardized a large number of performance tests designed to measure speed, extent, precision, and steadiness of movement,

^f Healy, William, and Bronner, Augusta F., *Delinquents and Criminals, Their Making and Unmaking*. New York: The Macmillan Company, 1926, p. 152.

^g Glueck, Sheldon, and Glueck, Eleanor T., *One Thousand Delinquents, Their Treatment by the Court and Clinic*. Cambridge, Mass.: Harvard University Press, 1934, pp. 102 f.

^h Wholey, Cornelius, "Psychiatric Report of Study of Psychopathic Inmates of a Penitentiary," *Journal of Criminal Law and Criminology*, May-June, 1937, 57.

ⁱ Bromberg, Walter, and Thompson, Charles B., "The Relation of Psychosis, Mental Defect, and Personality Traits in Crime," *Journal of Criminal Law and Criminology*, May-June, 1937, 70-89.

¹¹ Wallin, "Diagnostic Findings," pp. 190 f.

motor coordination, form discrimination, constructive ability, resolution, determination; ability to follow instructions, to plan, to analyze objective situations, and to adapt means to ends; general motor ability or psychomotor age; mechanical ability, mechanical aptitude; the amount of skill possessed in specialized lines of craftsmanship; and "will-temperament" or volitional pattern. Scores of so-called form board, construction, puzzle, maze, performance, and mechanical aptitude tests, and batteries or scales of tests such as the Pintner-Pater-son, the Arthur, the Cornell-Coxe, and the Merrill-Palmer are now available.

According to the power of judgment or common sense

Binet emphasized that one of the fundamental defects in the whole group of the feeble-minded was lack of "common sense" or good judgment. This criterion is indeed more fundamental than mere erudition or even the intelligence level, for no matter how ignorant or otherwise defective an individual may be, he cannot be considered mentally defective in the socio-economic sense if he shows good judgment in the practical workaday situations of life. Many individuals who cannot qualify in the standardized intelligence tests of the conventional type have enough acumen or practical sense to manage their affairs with passable success. Although no definite scale of tests has been devised by which to measure the soundness or degree of maturity of judgment (unless the general intelligence tests do this indirectly), nevertheless the individual's ability to analyze, understand, originate, infer, judge, and reason can be estimated and observed in many intelligence and performance tests, in many practical situations of life, and in a few tests specifically designed to measure initiative, judgment, and common sense. Thus, to refer to only two of the older nontechnical tests of "common sense," the suspected mental defective is told to sit down on a chair previously turned upside down, or to put on a coat previously

turned inside out, and notation is made of whether the child has enough sense to right the chair or adjust the coat before proceeding to carry out the request.

According to the power of attention

One of the earliest tests of the degree of trainability of mental defectives was the so-called "fixation test" in which the child's eyes were stimulated by a bright light in a darkened room. If, instead of staring vacantly into space, the child was able to fixate the stationary light, and especially to follow with the eyes the light when it was moved about, he was considered to be trainable and to be a fit candidate for a training institution.

P. Sollier classified defectives on the basis of degree of attention defect as (a) absolute idiots, in whom attention is completely absent, (b) simple idiots in whom it is feeble and difficult to elicit, and (c) imbeciles, in whom it is unstable.

Value of criterion. Although this simple test has been superseded by radically different tests of far greater diagnostic significance, the attention criterion is not without value. Defects of attention are prevalent among all grades of the mentally limited. There is generally a fair correspondence between the strength of voluntary attention and the degree of intelligence and trainability. In general the more pronounced the intelligence defect, the more difficult it is to secure and hold the attention. The lower grades of mental defectives are largely incapable of other than passive attention or attention based on organic needs. Voluntary attention is aroused and held with extreme difficulty. Attention constantly roves in idiots. Imbeciles attend voluntarily for only brief periods of time; they quickly tire, and are constantly distracted by all sorts of trivial occurrences. Their attention possesses little depth or persistence and little tendency toward spontaneous return after distraction. Morons are capable of voluntary attention, but cannot concentrate for long on abstractions. They are easily distracted but will spontane-

ously return to their assigned tasks. Backward and delinquent children are often listless and inattentive because they are disinterested in the subject matter. They often fatigue rapidly, but the fatigue may be due to boredom and is more apparent than real. The young infant makes little mental progress until the roving eyes can be fixed, and the attention caught. In fact, attention is coterminous, and almost synonymous, with consciousness. Wherever there is consciousness there is a modicum of attention. It is a good practice for every teacher to observe the characteristics of the child's attention: the readiness with which it can be aroused, its depth or the degree of absorption, its range, persistence, ease of distractibility, and tendency to return spontaneously after distraction. The teacher's first educational imperative is so to present the subject matter and arrange the learning situations as to attract and hold the pupil's attention. Little learning or educational advance occurs in a state of inattention. It is worth emphasizing a truism: the teacher does not get the child's attention merely by demanding it! She must earn it through animated presentation and skillful management of all the learning devices at her command. The inability of the child to concentrate or to sustain attention is a suspicious sign, suggestive of obstructed breathing (producing aprosexia, or inattention due to physical defects), or malnutrition, or mental deficiency, or nervous, emotional, and volitional instability, or mental conflicts, or similar defects and difficulties.

Limitations. Nevertheless, valuable as this criterion is, the correlation between degree of attention and grade of mentality or intelligence is not perfect. The neurasthenic works rapidly and energetically, possessing considerable depth of attention, but is quickly exhausted, possessing little persistence of attention. Although the neurasthenic may be highly intelligent, attention soon flags. The attention in many psychotics is very easily distracted, although there is little evidence of intellectual deterioration. Fatigue, lack of sleep,

lack of interest, emotional disturbances, malnutrition, obstructed breathing, and a stuffy atmosphere may induce inattention even in brilliant children. It is also possible that specific volitional or attention defects may exist without obvious intellectual defects.

*According to degree of speech development
or speech defectiveness*

Esquirol divided the mentally defective into three grades on the basis of speech ability: (1) the mutes, who cannot produce any sounds at all, (2) those who can utter only cries or monosyllables, and (3) those who can form words or short detached phrases or broken sentences.

In the classification of Binet and Simon the idiot cannot talk, the imbecile can speak but cannot communicate in writing, and the simple debile (a moron reaching a maximum mentality of nine years) can speak, read, and write.

According to Tredgold no idiots can form sentences, many cannot speak at all, and some can pronounce a few monosyllables. Imbeciles, although their vocabulary is very limited and their articulation frequently imperfect, can usually understand and speak short sentences. Although the morons have larger vocabularies and better speech, few can understand sentences that are complicated in construction.¹²

Makuen regards speech development and the ability to improve from speech training as one of the best diagnostic tests of mental defectiveness.

In the writer's experience absolute mutes are very rare among mental defectives; the best of the idiots can utter only a few short words, usually indistinctly; the best of the imbeciles, with marked poverty of ideas, can speak in short sentences or phrases, often indistinctly, and the best of the morons can express simple trains of thought, often quite distinctly, in complete simple sentences and often in fairly complicated complex sentences.

¹² Tredgold, *Mental Deficiency*, p. 145.

Facts showing relationship between speech imperfections and mental defectiveness. What is the nature of the facts that show the close relationship between delayed speech and speech defects and mental deficiency? The results of a few investigations are apropos.

Mead found that the average age when 25 sons and 25 daughters of Columbia University graduate students were able to "use a word intelligently" was 15.7 months, with an extreme range of from 9 to 25 months, whereas the median for 92 institutional cases of mental defectives (probably of the higher grades) was 34.2 months, with an extreme range of from 12 to 156 months.¹³

Lapage found that 61 high grade mental defectives began to talk at 1.8 years, and 50 low grade at 3.5 years, that 88 had good speech at 1.9 years, and that 64 had defective speech at 3.2 years.¹⁴

Among clinic examinees the writer found that 278 normal children (that is, not mentally defective, although some were backward) first used single words at the average age of 1 year (the upper limit being 4 years), 272 subnormal children at 1.8 years, and 164 mentally defective children at 2 years (with an upper limit of 7 years), the age being 1.6 years for the morons and 2.3 years for the imbeciles. The normals first used short phrases or sentences at 1.7 years (with an upper limit of 8 years), the subnormals at 2.6 years, and the mentally defective at 3 years (with an extreme of 11 years), the corresponding figures for the morons and imbeciles being 2.3 and 3.7. The city-wide survey revealed over nine times as many speech defects among the children in the special schools for the mentally defective (some of the children, however, were on the borderline or merely backward) and seven times as many among the children examined in the clinic (about 90 per cent of whom were more or less subnormal, and about

¹³ Mead, Cyrus D., "The Age of Walking and Talking in Relation to General Intelligence," *Pedagogical Seminary*, 1913, 460-484.

¹⁴ Lapage, C. Paget, *Feeble-mindedness in Children of School Age* (2d edition). London: Longmans, Green & Co., 1920.

35 per cent mentally defective), as were found among pupils in the regular elementary and high school grades. In the special schools 26.3 per cent of the pupils had speech defects (exclusive of minor imperfections among the imbeciles) as against 2.8 per cent in the regular elementary and high school grades. Of the clinic examinees (545 cases, Table V) 19.6 per cent were subject to some kind of speech defect or disorder; 52.8 per cent of these cases were classified as mental defectives. Among the subnormal clinic cases 81 per cent of the speech defects were articulation defects (inarticulate enunciation, lisping, lalling, and letter substitution) and only 11.2 per cent were cases of stuttering and stammering. The corresponding figures among the pupils in the regular grades are 57.1 per cent and 26.9 per cent.¹⁵

Value of criterion. Notable delay in the acquisition of speech, or defectiveness of articulation, or unintelligibility of enunciation constitutes a presumption of mental defect in the absence of an adequate specific cause. A child who cannot speak at six or seven, or one who is still talking baby talk, or speaking very indistinctly, lisping, or slurring badly is probably mentally defective unless the speech delay or defect can be traced to deafness, impaired hearing, motor aphasia, auditory aphasia (word deafness), paralysis of the speech muscles, organic defects,¹⁶ lack of opportunities for acquiring speech (social isolation), or emotional blockage. Speech may be inhibited (psychic mutism) because of emotional conflicts and thereby arouse a suspicion on the part of an examiner of mental defect.

¹⁵ *Report on Speech Defectives in the St. Louis Public Schools*. Annual Report of the Board of Education of the City of St. Louis, Missouri, for the year 1915-16, pp. 174-211; reprinted as "Speech Defective Children in a Large School System," *Miami University Bulletin*, XXV, 4: 1-45, 1926. "A Census of Speech Defectives among 89,057 Public School Pupils," *School and Society*, 1916, 215-216. "Diagnostic Findings," May-July, 1923, pp. 187 f.

¹⁶ For the case of a boy of nineteen who had not learned to talk because of tongue-tie, leading to a mistaken diagnosis of imbecility, who made normal educational and mental progress after the needed operation, see Makuen, G. Hudson, "Training of Speech as a Factor in Mental Development," *Bulletin of the American Academy of Medicine*, 1898, 3: 501-505.

ILLUSTRATIVE CASES: speech inhibitions produced by emotional blockage. Two Italian siblings were referred to the clinic because they "would not talk in school." The older brother, J. F., was in the fourth grade after five years in school, had a Stanford-Binet age (Form L) of 9-0 at the age of 10-3, I. Q. 88 and a Cornell-Coxe performance I. Q. of 106. His Wallin-Gilbert (Cutsforth) rating was grade I in reading, grade II in spelling, and grade III in written language and spelling. In the Monroe Diagnostic Reading Test the average reading grade was 1.7 and the spelling grade 2.7. The grade placement according to the Stanford Computation test was 3.7. The examiner found no difficulty in obtaining oral responses or in engaging the boy in conversation. This was also true for the younger brother, E. F., age 9-7, Stanford-Binet age (Form L) 7-10, I. Q. 82, who was in the third grade and had "never spoken a word in school for three years." In the Wallin-Gilbert scale he rated sub I in reading, II in written language and spelling, and III in arithmetic. His Arthur Performance age was 10.4, I. Q. 107, his Cornell-Coxe age was 13-0, I. Q. 140, and Goodenough Drawing age 12-0, I. Q. 121. Neither of these boys was mentally deficient. In performance ability they rated normal or better. Investigation seemed to show that the speech inhibition in J. F. was caused by the fact that he had been "made fun of in school because he talked Dago." The mother could not speak intelligible English and the father spoke "broken English." The younger child apparently "copied his brother" although his inhibition may have been deepened by the teacher's threat that "he would have to stay in after school if he talked in the class."

The following speech inhibition is traceable to a careless threat. I had a pupil, eight years of age, in the first grade who would not respond in school in any way, shape, or form. On her way with me to school she would talk eloquently. She was a very good conversationalist. Upon talking with her father I found that he had informed her that if she talked in school I would cut out her tongue. It took almost a year of coaxing, "hiring," and other devices to get her to talk. However, before the end of the year she had almost recovered from this "complex." She did fair work but not as good as she would have done had she not acquired this inhibition. She informs me that even now many years later this fear creeps over her once in a while.¹⁷

¹⁷ Wallin, *Minor Mental Maladjustments in Normal People*. Durham, N. C.: Duke University Press, 1939, pp. 78, 86, quoted from p. 180.

Although many exceptions exist to the generalization that speech defectiveness and speech delays vary with the degree of intelligence, exceptions also exist to the converse implication that a child is normal because he can talk glibly or fluently. Some imbeciles are exceedingly loquacious and possess amazing fertility of expression. Though they may be perfect chatterboxes, their speech often consists of inconsequential jabber, mechanical logorrhea (*logos*, word; *rhein*, to flow), or confabulations. They often deceive the unwary layman. To illustrate: the members of an audience of teachers at a demonstration of psychoclinical testing were invited to interrogate the examinee, a boy of about thirteen who could chatter very fluently and who gloried in the opportunity to talk. After he had prattled on with great gusto and fluency for a few minutes, the auditors were requested to jot down on slips of paper provisional diagnoses of his intelligence level. A few thought he was bright, a larger number rated him as normal, an equal number classed him as backward, and two or three diagnosed him as a moron. No one suspected that he was an imbecile with a Binet level between five and six years. Two simple questions regarding the number of fingers on each hand made it perfectly obvious that he was a mere "polly." He said he had 7 fingers on the right hand and 12 on the left. The same question during two earlier examinations brought forth different answers each time. The mere fact a child can talk volubly and facilely does not constitute proof that he is not an imbecile.

Putative "deprivation" types of mental defectiveness should be considered in connection with psychological classifications.

According to degree of sensory defect

The assumption is that slight sensory defects may lead to some degree of mental dullness and that complete deprivation of the use of the most important senses, vision and hearing, might produce mental deficiency. It is argued that a child might remain immature or undeveloped and eventually stag-

nate as permanently mentally deficient because of congenital or early acquired blindness or deafness which has not been overcome by appropriate remedial educational treatment. In other words, if the apparent mental defectiveness is due to sense deprivation, effective corrective educational treatment will remove the defect if it is begun early, but if the child is neglected unduly long, permanent secondary amentia might result. The marvelous extent to which the effects of sensory defects may be compensated for or overcome by the development of other sense organs is well known. Had Laura Bridgman and Helen Keller been sent to an institution for the mentally defective and been given merely the routine instruction provided in such institutions, they would probably have stagnated as mentally defective and have remained unknown to the world. If the sensory defect is superimposed upon a background of primary mental deficiency—if the child suffers from a double handicap—the educational possibilities are sharply circumscribed. But the programs of orthogenic education in schools or classes for the blind and the deaf have demonstrated that the large majority of these sensory defectives are intellectually potentially normal and some are intellectually brilliant, and that they are capable of making great educational progress with the aid of the teaching techniques devised in their behalf, such as finger reading of Braille for the blind and lip reading and speech for the deaf.

*According to degree of social deprivation
or isolation*

The assumption is that lack of cultural stimulation and of social and educational contacts may produce varying degrees of intellectual stagnation or deterioration from suppression of latent maturation or from "functional atrophy of the brain because of disuse." The amount of the intellectual arrest would vary with the extent and duration of the social neglect or isolation. Many so-called wild, wolf, savage, or feral chil-

dren who have been found living among wolves, bears, wild sheep, and oxen have been diagnosed as cases of mental atrophy through disuse, or "idiots through isolation." Itard so diagnosed the savage of Aveyron, an eleven- or twelve-year-old boy who was found in 1798 by huntsmen in the woods of Caune in the Department of Aveyron, in the southern part of France, roaming the forest unclothed in search of acorns and nuts, unable to speak, walking on all fours, drinking water while lying flat on the ground, and biting and scratching those who attempted to interfere with him. Itard, a physician and empiricist, or sensationalist (all knowledge or mental contents come from experience or sensations), thought the boy was merely wild and untaught and mentally arrested because of social and educational neglect, and believed that he could be greatly improved or restored to normality by exciting his nervous sensibility with varied and energetic stimuli, by supplying his mind with the raw impressions of ideas, by the imitative development of speech, by applying his mind to the satisfaction of his growing physical wants, by arousing new social needs, and by leading him to prefer social life.¹⁸ Partly because he wanted to demonstrate the truth of this theory and the correctness of empiricism, Itard undertook to lead the boy by gradual steps "from natural life to social life" and "from savagery to civilization." After a year of exceedingly arduous and devoted labor, Itard modified his methods, without having openly acknowledged that his diagnosis had been mistaken, and adopted methods more consonant with the needs of an idiot or low imbecile than a normal savage. After four years of the most systematic and painstaking course of sense training on record (supplemented by attempts to teach reading, writing, and to develop speech, comprehension of spoken language, and power to generalize) and of the individual (clinical) study of the educational needs of one child, Itard, in deep disappointment, dismissed

¹⁸ Itard is said to have generalized Pereire's touch training and specialized Rousseau's theories in his educational program.

his pupil precipitately because, at the onset of puberty, the boy broke out in a "wild storm of passion," and Itard felt that he had failed in his mission.

The most recent wolf children, two girls, Amala and Kamala, were rescued from a wolf den in a jungle near Midnapore, India, on October 17, 1920 and brought to an orphanage by their rescuers, where the attempt was made until their deaths to educate and civilize them.¹⁹ The older girl attained the mentality of a young child before she died at seventeen, being able to run simple errands and speak in sentences. (For an instance of extreme social deprivation in the United States, see p. 100.)

The University of Iowa environmentalists and the shifting I. Q. Based on the facts revealed by repeated Binet testings of the same children, a number of psychologists connected with the Iowa Child Welfare Research Station have recently attributed supreme importance to the influence of the child's early social and educational environment upon his subsequent intellectual development. Comparisons have been made of the course of the I. Q. curves of children domiciled for years in culturally unfavorable environments—underprivileged homes, an institution for the feeble-minded, a barren cottage in a state orphanage—and in favorable environments.

To refer briefly to one of the investigations, a three-year experiment on two groups of children in the Iowa Soldiers' Orphans' Home matched as to chronological age (a mean of 44 months), intelligence age, intelligence quotient (82 and 81, based on the Kuhlmann-Binet for the younger and Stanford-Binet for the older children), nutritional status, sex,

¹⁹ For a full account of this experiment and the most complete review of the extant writings on feral children and children isolated from human contacts in dark rooms, attics, or elsewhere see Singh, J. A. L., and Zingg, Robert M., *Wolf Children and Feral Man*. New York: Harper & Brothers, 1939; also Gesell, Arnold, *Wolf Child and Human Child*. New York: Harper & Brothers, 1942 (narrative interpretation of Kamala). On Itard's experiment see Itard, J. M. G., *Wild Boy of Aveyron*. New York: Century Company, 1935 (translated by George and Muriel Humphrey).

and length of residence in the institution. Not all children continued throughout the course of the experiment because some left the institution and new admissions were received. Thirty-five children (the control group) congregated in one cottage, in charge of an untrained matron, received no individual attention, had no play materials, and had little contact with life. One of these children lost 43 I. Q. points (from 103 to 60) in less than three years and a second child lost 37 points in the same time (from 98 to 61). A third child declined from 86 to 62, a loss of 24 points, and a fourth child moved from 83 to 60, a loss of 23 points. Twenty-six children with an average I. Q. of 90 lost 16 points (to 74) and deteriorated "perilously close to feeble-mindedness"²⁰ according to the report.

Fifty-nine children were afforded a rich program of constructive activities, directed games, musical experiences, stories, lunches, and rest periods in a preschool from eight to five o'clock. The average gain was 5 I. Q. points for those in residence 400 or more days while the control group lost 5 points. Of the children with I. Q.'s above 80 those in the preschool maintained their status during this period while the control group lost 16 I. Q. points. One child with an I. Q. of 98 at three and a half years rose to 167 at five (an increase of 69 points to the status of a potential genius), but had declined to 143 at the age of twelve. Another child with an I. Q. of 89 at three had gone to 149 at ten and a half (an increase of 60 I. Q.), but had receded to 132 at thirteen and a half. These extraordinary increases were attributed to the superior "environmental stimulation" of the preschool. Apparently the Binet I. Q. affords a measure of environmental stimulation valid only at the time the test is administered, rather than a measure of native ability. To quote, "there is now no escape from the fact that the I. Q.'s of children have

²⁰ Wellman, Beth L., "Our Changing Concepts of Intelligence," *Journal of Consulting Psychology*, July-August, 1938, 97-107; Skeels, Harold M., Updegraff, Ruth, Wellman, Beth L., and Williams, Harold M., *A Study of Environmental Stimulation: An Orphanage Preschool Project*. University of Iowa Studies in Child Welfare, No. 4, 1938, p. 15.

possibilities of change over a large portion of the I. Q. range from genius to feeble-mindedness. Children can and do change in test I. Q. from average to genius and from average to feeble-mindedness," depending upon the stimulating or nonstimulating character of the environment.

For a long time we have given lip service to the concept of feeble-mindedness from social isolation but, practically, the concept has remained a dead letter. The majority of specialists on mental defect do not even mention the possibility of mental defectiveness through social deprivation. Apparently the assumption has been accepted that environments so lacking in intellectual stimulation as to produce feeble-mindedness do not exist on the reality plane in this country or that feeble-mindedness engendered by a socially impoverished environment is a myth. Of many thousands of cases passing through the writer's clinics only four instances of serious mental retardation now come to mind which were ascribed purely to mental or educational neglect. The case files for these adolescents are not now at hand, but the I. Q.'s based on the 1911 Vineland Binet varied from about 60 to 72. Two unschooled sisters, who had been put to work in a mill in Florida at the age of six, were brought by a religious organization to Pittsburgh at the ages of ten and fourteen for an education and placed in a child welfare home. While testing mentally deficient, they did not present quite the symptom complex of the mental defective. The same can be said of a nineteen-year-old boy from the same state who had never been in school, who reached my clinic in St. Louis, referred from a first grade, at the age of eighteen when he had a Binet level of about nine and was diagnosed as a case of mental defect through cerebral atrophy superinduced by social and educational neglect; and of a fourteen- or fifteen-year-old girl, also without any schooling, from a backwoods mountain section in West Virginia. None of these would rate below the high grade defective or borderline level. Although few authorities have made any use of this concept, we are now told by the Iowa investigators that whether children of the same initial I. Q. are destined for feeble-mindedness or for genius depends almost solely upon the character of the environmental stimulation to which they are subjected.²¹

²¹ Wallin, "The Results of Multiple Binet Retestings of the Same Subjects: The Educational Implications of Variations of Test Performance," *Journal of Genetic Psychology*, 1940, 345-391; see also *Journal of Exceptional Children*, March, 1940, 211-222.

The sweeping conclusions reached by the Iowa school of psychologists reflect the views of the Watsonian school of behaviorists or environmentalists. Listen to John Watson, the American prince of behaviorism:

We have no real evidence of the inheritance of traits. I would feel perfectly confident in the ultimately favorable outcome of careful upbringing of a healthy, well-formed baby born of a long line of crooks, murderers, thieves, and prostitutes. . . . I should like to go one step further tonight and say, give me a dozen healthy infants, well-formed, and my own specified world to bring them up in and I'll guarantee to take any one at random and train him to become any type of specialist I might select—a doctor, lawyer, artist, merchant-chief, and, yes, even into beggarman and thief, regardless of his talents, penchants, tendencies, abilities, the vocations and race of his ancestors.²²

The Iowa Child Welfare Research Station workers seem to have taken their cues from Watson's apotheosis of the environment: environmental stimulation is the alpha and the omega of child growth and development.

A parallel experiment in the Chicago special classes. More recently a somewhat comparable experiment with equally astonishing results has been reported by Schmidt,²³ based upon a study of 322 adolescents, 220 girls and 102 boys, ages twelve to fourteen, "competently classified as feeble-minded," by "highly trained, competent psychologists," and assigned to five centers for such children located in elementary schools in

²² Watson, John B., "What the Nursery Has to Say About Instincts," *Pedagogical Seminary and Journal of Genetic Psychology*, June, 1925, 301 f.

²³ Schmidt, Bernardine G., "Changes in Personal, Social, and Intellectual Behavior of Children Originally Classified as Feeble-minded," *Psychological Monographs*, No. 281, 1946. See also the following brief summaries: "The Rehabilitation of Feeble-Minded Adolescents," *School and Society*, December, 1945, 409-412; "Changes in Behavior of Originally Feeble-minded Children," *Journal of Exceptional Children*, December, 1947, 67-72, 94.

The experiment has been widely publicized in the popular press as a demonstration that feeble-mindedness is curable. See e.g., Stern, Edith, "Feeble-Minded Children Can Be Cured," *Woman's Home Companion*, September, 1947; Clark, Blake, "They Are Feeble-Minded No Longer," *The Reader's Digest*, September, 1947, 111-115.

Chicago. Two hundred and fifty-four boys and girls attended three "experimental centers" and 68 girls two "control centers." The children were equated, we are told, in the experimental and control groups with respect to chronological age, I. Q.'s, time enrolled in school, and achievement and socio-economic status. Batteries of objective psychological, achievement, and personality tests were administered by the investigator at the outset of the experiment and at eighteen-month intervals thereafter during the three-year in-service period in which the children attended the special classes and during the five-year post-school period. The tests included the 1916 Stanford-Binet scale for all groups except one, which was given Form L, the Stanford Achievement Battery, Forms V and W, the Bernreuter Personality Inventory, Pintner's Aspects of Personality, the Willoughby Emotional Maturity Scale, the Vineland Social Maturity Scale, and the Detroit Adjustment Inventory. Concurrently, observational studies were also made of the pupils' personal, social, and intellectual behavior patterns, in and out of school, and of their post-school adjustment in the family and in the community. Records of employment and delinquency were compiled, and studies were made of the cultural, social, and economic conditions in the home environment.

The control centers, which proceeded upon the assumption of "limited ability to learn" on the part of the pupils assigned, "provided a curriculum which paralleled that of the regular elementary school in kind at an extremely slow rate with emphasis on quantities of orthodox drill and a bulk of time devoted to pure handwork." The program in the experimental centers, on the other hand, was planned, not only to improve academic and manipulative skills, but to "decrease nervous tensions," to "remove emotional blockages," to overcome emotional instability, to develop "emotional and social maturation," to further "social interaction," to improve work and study habits, to develop "self-confidence, self-reliance, and a sense of personal worth," to emancipate the child from

parental dependence, and to train him to meet conflict situations directly instead of by some kind of escape, substitute, or defense mechanism. The instructional techniques emphasized group planning of the units employed, suggested by the pupils, group experiences, practical community activity projects, creative and manipulative arts, household mechanics, personal grooming and etiquette, health development, leisure-time activities, self-government, and the like. These objectives and instructional procedures are in harmony with those of progressive educationists. No assumption was made in these centers regarding the inability to learn. The program was adjusted to meet the needs of each child as they were revealed in the home and in the school. None of the teachers was a college graduate. The teachers merely had about six months of special training. The staff included a shop teacher in each center and teachers of music, recreation, and health.

The results of the experiment in these centers may be summarized briefly from the detailed record as follows: the mean grade achievement based on the Stanford Achievement Battery rose in reading from an initial 1.4 to 5.5 at the end of the three-year period, a gain of 4.1; and in arithmetic from 1.3 to 4.9, a gain of 3.6. The mean Vineland S. Q. (social quotient) rose from an initial 59.4 to 91.8 at the end of the in-school period and to 107.2 at the end of the experiment, a rise of 47.8. The mean Binet I. Q. rose from 52.1 at the start to 71.6 at the end of the three-year period and to 89.3 at the end of the post-school period, a gain of 37.2 points (and not 40.7 as stated by the investigator, p. 117). At the end of seven and a half years, 80.7 per cent of the 100 then retested had gained over 30 I. Q.'s, 59.6 per cent over 40, and 29.3 per cent 50 or more. The extreme individual range was from a drop of 4 points to an increase of 71. At the start 53 per cent of the children had been classified as imbeciles and low morons "on the basis of intelligence," but at the end only 2.7 per cent had I. Q.'s between 50 and 57; 86.4 per cent ranked as dull or normal; 59.7 per cent as low or high normals; and only

7.5 per cent "were still feeble-minded." At the end of the eight years, 27.2 per cent had finished high school and 5.1 per cent (over 13 per cent, according to p. 68) had continued on after high school. At the end of the investigation, 83.4 per cent were regularly employed, with an average service record of over three years, and 5.9 per cent were in military service. Only one was "permanently idle," although eight of the pupils had been excluded as ineducable. Thirty-six and one-tenth per cent were employed in unskilled and semi-skilled jobs; 28.1 per cent in skilled services; and 35.6 per cent in clerical work. Four persons held professional positions. The mean weekly wage for the boys was \$28 (from \$13 to \$84), and for girls, \$27 (from \$10 to \$60). All of the 15 married girls and 5 married boys were "maintaining financially independent family units" (p. 75). None of the children held court records while attending the special centers. "By the end of the study, the average adjustment of the total experimental group was equal to that of the average adult."

Although far fewer data are supplied on the control group, at the close of the in-school period the composite grade level was 1.9 as against 5.0 for the experimental group (Table 75). None earned a certificate on leaving the special center, as compared with 23 girls in the experimental center. None continued in school after withdrawal from the center. Only 13 per cent had been employed, exclusively in unskilled jobs, at some time during the post-school period, for an average of three months, at a mean weekly wage of \$8.75. Eight of the girls had illegitimate children (contrasted with one in the experimental group), seven of them being dependent upon public assistance. Twenty-three and five-tenths per cent had been in the juvenile court, as compared with one from the experimental group (Table 76). During a five-year period the control groups registered a mean drop of 3.6 Binet I. Q. Individual losses reached 20 and 22 points. Only four showed any improvement, the largest gain being four points.

Were the results of this investigation accepted at face value.

the inevitable implications would seem to be that: the environment is the be-all and end-all of human development; the diagnosis of feeble-mindedness cannot be made on the results of intelligence, achievement, or social maturity tests, irrespective of the emotional condition and cultural background of the examinee and before his intellectual processes have been liberated from the thralldom of emotional inhibitions, blockages, and conflicts;²⁴ the feelings of satisfactory achievement, emotional security, and self-confidence are basic for the liberation of the creative impulses, for mental growth, and for social and educational achievement; much so-called mental defectiveness is really pseudo-feeble-mindedness,²⁵ the result of misdiagnosis, and is, therefore, purely functional. Although these conclusions are not without merit, and may win qualified acceptance, many experienced students of mental defect will, no doubt, remain adamant in their conviction that genuine mental defectiveness is organic in nature, whether the result of constitutional defect or of secondary injury, and cannot be eradicated merely by cultural or educational processes of stimulation, no matter how much intellectual and social improvement may be thus produced. Although Schmidt concludes "that a majority of children originally classified as feeble-minded can grow to be mentally competent, well-adjusted members of a democratic society, if their educational program is so planned to meet [*sic*] their emotional as well as academic needs while in school and to prepare them for social and vocational competency in their post-school years," nevertheless, she concedes that "the multiplicity of factors interacting in the adjustment process makes difficult any determination of causal relationships in the development of social competency" (p. 119), and that the "iso-

²⁴ Years ago the theory was advanced that some cases of feeble-mindedness might be produced by emotional repressions by Clark, L. Pierce, *The Nature and Treatment of Amentia*. Baltimore: William Wood & Company, 1933.

²⁵ Arthur, N. Grace, "Pseudo-feeble-mindedness," *American Journal of Mental Deficiency*, October, 1947, 137-142.

"Pseudo-feeble-mindedness," *The Nervous Child*, October, 1948, 363-445 (a symposium).

lation of the crucial factors in this program must wait on further research."

CRITICISM. The basic issues involved in the evaluation of this investigation are: what proportion of the children in the groups were actually feeble-minded in the accepted connotation of that word? and how accurate are the reported test findings? In the attempt to supply reliable answers to these questions only a limited amount of factual information is available at this time.

Kirk in his on-the-spot investigation of this research²⁶ states that a "perusal of the records in the files of the Bureau of Child Study showed that the psychologists did not classify any of the children as 'feeble-minded,' as claimed by Dr. Schmidt." "The classification 'feeble-minded' is her own designation." Nothing in the record suggests that the diagnoses of feeble-mindedness were based on complete case study procedures (anamnesis, personal and family histories, medical examinations, etc.). The study is barren of individual case histories (with the possible exception of five twins) that can be traced from identifiable symbols. Facts have shown beyond cavil that an arbitrary intelligence criterion (usually a Binet I. Q. below 70) cannot be used as a short cut for the diagnosis of feeble-mindedness in its socio-legal connotation, that is, from the standpoint of practical socio-economic efficiency adequate for self-maintenance at a marginal level. The assertion that the children were "competently classified as feeble-minded" by "highly trained, competent psychologists" is, therefore, without justification.

In commenting on the publicized reports that these groups contained children who "ranged in I. Q. down through the 20's with the group median in the 50's," Grace Munson, the director of the Bureau of Child Study in the Chicago schools, makes the following statement: "This of course is entirely in-

²⁶ Kirk, Samuel A., "An Evaluation of the Study by Bernardine G. Schmidt: Entitled: 'Changes in Personal, Social and Intellectual Behavior of Children Originally Classified as Feeble-minded,'" *Psychological Bulletin*, July, 1948, 321-333. See also Schmidt, Bernardine G., "A Reply," *ibid.*, 334-343.

correct since we do not place in our ungraded divisions children with I. Q.'s in the 20's. Furthermore no ungraded division in the city would have its median I. Q. in the 50's." ²⁷ This statement is supported by the facts supplied by Kirk. The average Binet I. Q.'s for children recommended for the ungraded division for the years 1937 to 1940 varied from 68.0 to 69.1, with a range of from 30-39 (with one exception) to "80 or above." In the tabulation of the Binet I. Q.'s (based on the last test results before admission) of 32 children enrolled in 1936-37 in two of the groups, he found the median I. Q. to be 69 in one group and 64 in the other, giving an average of 67. The ranges were from 27 (for a child eliminated after 42 days of attendance) to 92 in one group and from 41 to 85 in the other. These Binet findings correspond closely with the distribution in the entire system of ungraded classes. Over 50 per cent in one class and 29 per cent in the other class had I. Q.'s above 69, the highest I. Q. reported by Schmidt.

Is not the conclusion inescapable that a large ratio (perhaps 50 per cent or more) of the children included in this investigation cannot be legitimately classified as feeble-minded and that the categorical pronouncement that "feeble-minded children can be cured" by processes of education has not been demonstrated?

Three further comments on the Chicago investigation are apropos:

(1) Some of the evaluations are doubtless based on subjective impressions. Subjective elements may even influence scoring of tests, such as the Vineland Social Maturity, the Binet, the Bernreuter, and others. To what extent a favorable mind set on the part of the examiner may have tilted the scales for borderline or uncertain responses in a favorable direction and thereby have produced spurious increases cannot be determined from the record. It is highly questionable whether children whose "average reading achievement for the total

²⁷ Munson, Grace, "Analysis of Statistics on Individual Psychological Case Studies," *American Journal of Mental Deficiency*, 1947, 614.

group was just below that of the child beginning the last half of the first grade" would be able to do the Bernreuter at all, which requires high school reading ability. How was the Bernreuter administered? In view of the damaging disclosures already made, all of the tests administered by the author herself, allegedly every 18 months, should be thoroughly authenticated.

(2) The very favorable occupational record of the experimental group is probably due to a number of factors other than the efficiency of the instruction. (a) Many of the children were merely backward and some were doubtless normal at the time of admission and not actual cases of feeble-mindedness. (b) The experiment apparently began in November, 1935, while the depression was still in full swing, and ended (December, 1943) in the early part of the postwar inflation, when jobs at inflated wages were available for the asking by anyone, irrespective of ability or competency. This factor doubtless accounts to some extent for the high occupational success of many of these adolescents. (c) Occupational information was freely supplied to the experimental group. Interviewers from the employment offices visited the schools. From the published account it is, however, not possible to determine to what extent the favorable employment records are due to this factor or to the maintenance of an efficient placement service.

(3) In a later somewhat similar study based on the Binet retests of 107 boys and girls at an average interval of 3-9 years from the Des Moines special classes, Hill was unable to confirm the Schmidt findings. Of the children retested 72 per cent remained "relatively static" within a variation of -7 and $+7$. The extreme deviates were equally distributed between those who lost and those who gained. The gains in some cases were presumed to be due to changes of attitude and various extraneous factors.²⁸

²⁸ Hill, Arthur S., "Does Special Education Result in Improved Intelligence for the Slow Learners?" *Journal of Exceptional Children*, 1948, 207-213, 224.

So far as the University of Iowa investigations are concerned, the conclusions have been vigorously assailed by a number of writers, particularly by McNemar, who has branded the "methodological and statistical inadequacies" of the investigation a species of "statistical jugglery" that gives a "nightmare to statisticians." In his opinion "differences of rapport need only be invoked to explain slight, statistically insignificant findings." He regards much of the supposed evidence for the environmental influences on the I. Q.'s as "entirely nonexistent." "Insignificant findings in favor of the environmental viewpoint have been constantly played up while the contrary findings have been ignored. . . . A large part of such gains and losses is due to errors of measurement. . . . Hasty promulgation of unverified and largely invalid research results" have been based on the "dramatic use of selected cases, falsely claimed to be typical."²⁹

Brief summary of other relevant studies with less spectacular positive findings or with negative outcomes bearing upon the improvement of intelligence from the placement of identical twins, foster children, and other matched groups of children in superior home and school environments. To arrive at a sound evaluation of the previous studies cited abstracts are given of other investigations, mostly from earlier dates, of identical twins, foster children, and other groups of school children who have been removed from inferior environments and placed in culturally and educationally superior ones. The reported findings of these studies at their best have been far less dramatic. The contradictory character of some of these studies is emphasized by the fact that some of them have yielded purely negative results. In this brief excursus only

²⁹ McNemar, Quinn, "A Critical Examination of the University of Iowa Studies of Environmental Influences Upon the I. Q.," *Psychological Bulletin*, February, 1940, 63-111; also "More on the Iowa I. Q. Studies," *Journal of Psychology*, October, 1940, 237-240; "Note on Wellman's Re-Analysis of I. Q. Changes of Orphanage Preschool Children," *Journal of Genetic Psychology*, December, 1945, 215-219. For the reply see Wellman, Skeels, and Skodak, "Review of McNemar's Critical Examination of Iowa Studies," *Psychological Bulletin*, 1940, 93-111.

the most important phases of some of the better known investigations are reviewed.

Studies of identical twins are supposed to yield unique opportunities for the study of the relative contributions of nature and nurture. Identical twins are the product of the same egg fertilized by a single sperm and should therefore possess the same heredity, aside from such variations as may be produced before birth by differential reactions to indeterminate differences in the maternal environment. However, in the absence of knowledge of the circumstances of the birth (one amniotic sac) it is not possible to determine with exactitude whether or not twin offspring are identical or fraternal. There are all degrees of resemblance in physical and psychic make-up between twins, and it has been shown that twins at first identified as identical twins on the basis of close resemblance were later found to have been fraternal twins (the products of two ova). But, assuming the accuracy of the diagnosis, the rearing of identical twins in different home environments has been accepted by geneticists as a crucial method of measuring the degree of modification of the genetic constitution that can be wrought by differences in the physical and cultural environment.

One of the most exhaustive investigations of cultural environmental impacts was that conducted by Newman, Freeman, and Holzinger on 50 identical twins and 50 fraternal twins reared together and 19 pairs of identical twins separated during the first year and reared apart for many years, often in very different surroundings. The twins came from the Chicago area. On the basis of numerous physical, psychological, and educational tests, the investigators reached the conclusion that "differences in the educational and social environment produce undeniable differences in intelligence and scholastic achievement as measured by our tests." The correlation of the Stanford-Binet I. Q. was .91 for the identical twins reared together but only .67 for the separated twins and .64 for the fraternal twins. (The correlation between ordi-

nary siblings is usually given as .50, sometimes referred to as the "coefficient of heredity.") The average difference in Binet I. Q.'s among the identical twins was 8.21 for the separated as compared with only 5.3 for those educated together.³⁰ The authors assume that the greater similarity of those reared together is due to the greater similarity of the common environment, although, obviously, the psychic environment and atmosphere might be dissimilar for children who grow up in the same home. The parents' reaction patterns are not always the same; they often treat different children in varying ways. Nor do different children react in just the same ways to similar or differing parental behavior manifestations.

Several years earlier Freeman, Holzinger, and Mitchell gave the Stanford-Binet (and also a performance test) to groups of dependent legitimate and illegitimate children in Illinois who were placed in foster homes.³¹ The foster homes were rated with respect to material resources, culture, the social activity and schooling of the foster parents and their performance in a vocabulary test and the higher form of the Otis Self-Administering Test of Mental Ability, and the occupation of the father. For 74 children in the preschool group the Binet I. Q.'s increased from a mean of 91.2 to 93.7 (at the age of 12-2) after four years in the superior foster homes, a gain of 2.5, which is interpreted as 7.5 points because of the drop in I. Q. in the Stanford-Binet from 111 at the age of five to 98 in age fourteen for the children on whom the scale was standardized. Thirty-three children in the better homes gained 5.3 I. Q. points, or 10.4 points corrected for age (the correction is

³⁰ Newman, Horatio H., Freeman, Frank N., and Holzinger, Karl J., *Twins: A Study of Heredity and Environment*. Chicago: University of Chicago Press, 1937.

³¹ Freeman, Frank N., Holzinger, Karl J., and Mitchell, Blythe C., "The Influence of the Environment on the Intelligence, School Achievement, and Conduct of Foster Children," *Nature and Nurture, Their Influence Upon Intelligence*. Twenty-Seventh Yearbook of the National Society for the Study of Education, Bloomington, Ill.: Public School Publishing Company, 1928. Part I. pp. 103-217.

regarded as too liberal by Merriman),³² whereas 41 children placed in the poorer homes gained only 0.1, or 5.0 points corrected for age. The younger children (less than 12-4) gained 5.2 points and the older ones (12-4 and over) lost 0.4. The correlation for 125 pairs of twins after they had been separated in different foster homes several years on the average was only .32 as compared with the accepted hereditary coefficient of .50. Those reared in the more similar homes were more alike (r , .39 for 62 pairs) than those reared in widely differing homes (r , .28 for 63 pairs). The mean I. Q. of 120 children with at least one mentally defective parent was 92.9. The mean I. Q. of these children who remained at home until at least five was 87.2, whereas those removed before five had a mean of 95.1. The mean I. Q. of 26 children both of whose parents were reported to be "definitely mentally defective" (according to estimates by home placement investigators) was 81.2; for the late removals 78.1; and for the early removals 86.0. The authors, who discount any selection on the part of the foster parents on the basis of intelligence, conclude that a "significant improvement in intelligence is associated with the type of foster home in which the children are placed." However, the mean improvement does not greatly exceed the extent of the usual retest variability.

Similar results were recorded in a parallel investigation carried out about the same time by Burks in California. The Stanford-Binet and other tests were administered to 214 children placed in foster homes before the age of twelve months, 71.8 per cent of whom were illegitimate and 6.9 per cent foundlings abandoned on doorsteps or elsewhere (probably also illegitimate). The children, from five to fourteen years of age at the time of the investigation, homogeneous as to race, educational opportunities, health, and physique, were placed with white, English-speaking, non-Jewish married couples living together harmoniously in three municipal areas in Cali-

³² Merriman, Curtis, "The Intellectual Resemblance of Twins," *Psychological Monographs*, 1924, 33: 331.

fornia. The Binet test was also given to the foster parents. The control children (105 cases selected from the school files) were matched with the experimental group as to age, sex, and race, and the type of neighborhood and occupation of the father. The mean intelligence ages (16 to 17), cultural index, and occupational status were about the same for the foster and true parents. It was considered that both groups of parents (the foster and the true) were superior to the average of parents of unselected progeny. The mean I. Q.'s were for the foster children, 107.4, and for the controls, 115.4 at the same mean chronological age of 8.2. The main conclusions from the study were that the improvement from favorable cultural environments was about the same as that revealed in the Chicago study, a mean of five or six I. Q. points. Burks attributes 17 per cent of the I. Q. variance to the home environment, with a maximum of 20 I. Q.'s or less from the best home environments; and about 33 per cent to parental intelligence alone. The total contribution of heredity "probably is not far from 75 or 80 per cent."³³

In the Wayne County Training School in Michigan 16 boys of the endogenous type from fifteen to eighteen years of age, chosen from a "self-determining cottage where they managed their own affairs," were provided with a stimulating school environment which encouraged "ingenuity, initiative, and original planning," and the solution of their own problems. Stanford-Binet retests at an average interval of one and a half years showed an average increase of 10.1 I. Q. points (from 66.3 to 76.4), with only one negative change (-3 I. Q.) which, perhaps, is about twice the variability without specific environmental stimulation. During the preceding year the average increase for the same group was only 2.3 I. Q. Another group of 26 boys of comparable chronological ages who were attending the regular school, which, however, was well

³³ Burks, Barbara S., "The Relative Influence of Nature and Nurture Upon Mental Development: A Comparative Study of Foster Parent-Foster Child Resemblance and True Parent-True Child Resemblance," *Twenty-Seventh Yearbook*, Part I, pp. 219-321.

supplied with materials for vocational training but which followed more formal procedures and placed the emphasis on memory work and automatization, registered a mean increase of only 1.9 points. The experimenter's conclusion was that the "rate of mental growth can be increased through specific programs of stimulation" for both younger and older children.³⁴

Contrasting with the positive findings cited above are the negative results of the following investigations. Sixty-four native American girls in Pennsylvania were removed from bad home surroundings and placed in cottages in two superior residential schools (one for delinquent girls) where they were afforded a large measure of freedom, the best type of modern education, an enriched curriculum, the kind of "social experience a child receives in an excellent family," and experience with real neighbors and friends. The chronological ages of the girls varied from four to thirteen. The first test (Stanford-Binet) was administered not later than four months after admission. The mean interval between the first and second tests was one year four months for one group and one year six months for the other one. The I. Q. changes, ranging from +20 to -21, with an average of +4.5, were about the same as the variations found in different investigations for unselected groups. The authors conclude that they found "no appreciable effect of environmental change upon the intelligence quotient. Such differences as are found may well be due to accidental factors."³⁵

Two groups of children in the New York City Lincoln School, comparable with respect to chronological age, sex ratio, superior economic and social status, selected at random from the standpoint of racial representation, 41 of whom had

³⁴ Kephart, Newell C., "The Effect of a Highly Specialized Program Upon the I. Q. in High-Grade Mentally Deficient Boys," *Journal of Psycho-Asthenics*, 1938-1939, No. 1, June, p. 216.

³⁵ Rogers, Agnes L., Durling, Dorothy, and McBride, Katherine. "The Effect on the Intelligence Quotient of Change from a Poor to a Good Environment," *Twenty-Seventh Yearbook*, Part I, pp. 323-331.

attended nursery school or kindergarten four months or more before the first Binet test was given, and 48 of whom had not attended school prior to the first Binet (although many probably had had nurses and governesses), were retested at an average interval of 18 months. The mean Binet I.Q. at the first test was 113.9 for those without schooling and 119.6 for those with schooling, a difference of 5.7. At the second test the corresponding I. Q.'s were 117.2 and 118.9, a difference of only 1.7. The difference between the latter means is a little less than twice as large as the P. E. of the difference (a difference to be statistically reliable should be four times as large). Although the extreme gains reached 29 I. Q., the extreme losses were almost as large (-25). Hildreth's conclusion was that "no real difference is found in the intelligence quotients of the two groups," both of whom were, of course, highly selected.³⁶

Page gave the Stanford-Binet Form L to 72 Rochester, New York children, about half of whom were of Italian extraction, enrolled in the kindergarten to the fifth grade, who had attended a full-day nursery school an average of 280 days (from 125 to 525 days) and also to their next older siblings who had not attended nursery school. The mean age of the nursery group was 6.96 and of the controls 7.10. Whether there was an equal distribution of the sexes is not stated. No direct relationship was discerned "between the number of days of attendance and subsequent I. Q. sibling superiority," or "favorable relationship between duration of nursery school attendance and subsequent I. Q. advantage." In fact, "the correlation coefficient between number of days of preschool attendance and I. Q. superiority of nursery children over nonattending siblings was -17 ."³⁷

³⁶ Hildreth, Gertrude, "The Effect of School Environment Upon Stanford-Binet Tests of Young Children," *Twenty-Seventh Yearbook*, pp. 255-359.

³⁷ Page, James D., "The Effect of Nursery School Attendance Upon Subsequent I. Q.," *Journal of Psychology*, October, 1940, 221-230 (contains a tabular summary of prior investigations). For summaries of numerous similar studies by different investigators, most of them yielding similar negative

One line of evidence that is favorable to the assumption that the basic core of the individual's intellectual endowment is determined by heredity rather than by environmental impacts should not be overlooked. It is the demonstrated resemblance of the intelligence test scores of the mid-parent (parental average) and the mid-child (mean of the siblings in the family), whether the parents are of average, inferior, or superior ability. On the basis of the Stanford-Binet (and the Army Alpha given to those above ten) Jones found a correlation of 0.69 between the mid-parent and the mid-child.³⁸ Cattell, using the Cattell nonverbal group and individual intelligence tests (purported to be culture-free) found a similar correlation of 0.73. Twenty parents with a mean I. Q. of 148.3 (corrected) had 23 children with a mean I. Q. of 147.0, and 20 parents with a mean I. Q. of 67.6 had 32 children with a mean I. Q. of 77.0.³⁹ In both instances the children showed a slight tendency of approximation toward the mean, a theory that has won some acceptance.

Wider implications of the experimental findings. The glaring discrepancies in the experimental findings and the questionable nature of some of the extreme positive outcomes lend little support for the smug and satisfying belief that the environment is omnipotent in the development of intelligence and that heredity counts for naught. The flimsy evidence on which such a belief is buttressed will not greatly modify the convictions of the hard-headed practical educationists and therapists who have spent their lives in the effort to provide mentally deficient children with the best possible educational

results, see The Thirty-Ninth Yearbook of the National Society for the Study of Education, *Intelligence: Its Nature and Nurture*, Part II, "Original Studies and Experiments," 1940, pp. 3-399. Part I, "Comparative and Critical Exposition," contains further summaries of studies of twins, and various environmental factors, pp. 201-204, 235-255, 405-442.

³⁸ Jones, Harold E., "First Study of Parent-Child Resemblance in Intelligence," Twenty-Seventh Yearbook, I, pp. 61-72.

³⁹ Cattell, Raymond B., "Is National Intelligence Declining?" *The Eugenics Review*, 1936, 28: 181-203; also "Human Fertility Trends Upon Distribution of Intelligence," Thirty-Ninth Yearbook, I, pp. 221-223.

facilities that modern school science affords, but who have been unable, figuratively speaking, to "transform sow's ears into silken purses." That the I. Q.'s of verbal tests like the Binet are influenced by cultural and educational impacts was freely conceded by pioneers in the Binet testing movement and is doubted by few competent psychoclinicians today. The only question in dispute is the amount of permanent improvement in the individual's intrinsic ability that can be produced by environmental stimulation—physical, educational, cultural, psychological. In the opinion of most students of genetics, native or constitutional limitations set boundaries to the extent of the permanent elevation of the general ability level that can be obtained from the improvement of environmental factors. A heightened level of functioning can probably be produced by unusual, excessive, or long-continued stimulation, but such "hot-house" increments may prove temporary, and the individual may eventually regress to his real or inherent ability level.

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Value of criterion. Theory and wishful thinking should not be permitted to interfere with sound educational practice. In spite of the serious doubts engendered in the minds of an army of physicians, psychologists, educators, and sociologists by a century and a half of scientific study of the causation, amelioration, cure, and elevation of mental defectiveness, the cautious student will strive to maintain an open, unprejudiced mind on this basically important question, supposedly settled once and for all many years ago but projected into the forefront of current issues in psychogenesis by the experiments on mental rehabilitation of the last decade. The alleged cure of feeble-mindedness by "environmental stimulation" of a cultural and educational character and by the release of the individual's latent intellectual potentialities by

emotional catharsis or therapy has brought new data to the forefront. Though the author is frankly sceptical regarding many of the claims of the restoration of mental defectives to mental and occupational normality, facts have shown incontestably that many school laggards and repeaters are capable of normal mental growth and development and can achieve satisfactory, and even pronounced, academic success when they are provided with vitally motivated and integrated instruction adapted to their individual requirements and providing adequate opportunities for the exercise and development of their initiative, resourcefulness, and individuality. Effectively organized teaching based upon the child's felt needs and interests—diagnostic, corrective, remedial, developmental, or concrete instruction, as needed—has made it unnecessary to transfer many dull and backward children to special classes.

All of the individual criteria discussed above possess varying degrees of relevancy to the determination and classification of mental defectiveness. According to our traditional concepts they are subsidiary in value to the more comprehensive, generic criterion of serious lack of general intelligence or of general capacity.

According to the degree of defect in general intelligence

Classifications based upon lack of general ability or upon general intellectual deficiency have almost superseded all others since the development of the so-called graded scales of intelligence during the first decade of the present century. The concept of a fundamental lack of general intelligence in mental defectives was probably presupposed or implied in the classifications of some of the older writers. Thus Esquirol (1828) pointed out that an idiot was incapable of acquiring the knowledge of a person of his own age under equally favorable external conditions. P. M. Duncan and W. Millard (1866) compared the "mental gifts" of the "mentally defective" with "those of children of perfect mind at younger ages," and J. L. Down (1887), in order to arrive at a conception of

the "mental condition" of the mentally defective child, "put back the age . . . in imagination . . . two or more years." But in the case of an idiot "no amount of imaginary antedated age" corresponds to "the present condition of the child." The mental ratings made were, however, largely offhand and subjective, representing the personal opinion of the individual examiner. No definite objective standards of measurement of general intellectual ability or normative age standards of mental development of typical children were available. The subjective judgments were probably based on medical (physical) findings, general appearance, facial features (that is, a vacuous, stupid appearance), behavior characteristics, success or failure in school work, or the grade standard reached in school.

The Binet tests. Alfred Binet, distinguished French psychologist, and Theo. Simon, French psychiatrist, made a unique contribution to the methodology of ascertaining the degree of mental deficiency when they constructed an age scale of intelligence, which appeared in preliminary form in 1905,⁴⁰ and in improved revisions in 1908 and 1911. Numerous revisions and extensions of this scale have since appeared in rapid succession in many lands, and some form of the scale is now in general use in the schools, courts, or institutions in almost every civilized country.

What is the nature of the Binet scale? What is its purpose? What does it consist of? Briefly, the 1937 Stanford version of the scale is composed of a series of varied objective tests arranged in age steps, six tests in each age with one exception, in an increasing order of difficulty from age II to Superior Adult III. The tests are designed to explore many aspects of "general intelligence." To illustrate from the seven-year tests in Form M,⁴¹ all very simple and easy to administer:

⁴⁰ A brief scale by S. de Sanctis appeared the same year.

⁴¹ Terman and Merrill, *Measuring Intelligence*. Boston: Houghton Mifflin Company, 1937, pp. 156 f. Quoted with the publisher's permission.

Test I, knowledge of number of fingers: "How many fingers have I on one hand? How many on the other? How many on both hands?" (All three correct without counting.)

Test II, memory for sentences: "Listen; be sure to say exactly what I say. 'Betty has made a pretty dress for her doll out of blue ribbon.'" (One of two sentences without error.)

Test III, picture absurdities: The child is shown in order three pictures, a man sawing off a tree limb on which he is sitting, a man with a bunch of books under his arm weighing himself on automatic scales, and a cat and five mice consorting peacefully together, and is asked as each is shown: "What's funny (foolish) about that picture?"

Test IV, repeating three digits reversed: "I am going to say some numbers, and I want you to say them backwards. For example, if I should say 5-1-4, you would say 4-1-5. Ready now: listen carefully and be sure to say the numbers backward. 2-9-5; 8-1-6; 4-7-3." (One correct after a single reading.)

The fifth test requires the child to construct a sentence containing three supplied words (horse, bigger, dog) and the sixth requires the child to tell how many taps were made by the examiner with a fountain pen cap, the series consisting of 7, 5, and 8 taps.

The testing proceeds from the basal age (in which all the tests are passed) to the age in which all the tests are failed. The intelligence age and I. Q. are determined from the total score (in months) in relation to the child's life age.

The Binet tests and similar psychometric tests enable the examiner to measure intelligence by a standardized scale of measurement. The tests, however, do not administer themselves like the indicator on a pair of spring scales. They cannot be properly administered without trained skill and clinical insight. They must be accurately administered to yield valid results.

It should be emphasized that the Binet scale is primarily a scale of verbal intelligence although it contains some performance tests. It should be supplemented by batteries of performance tests, particularly in the case of nonreaders, the

linguistically handicapped, and speech defectives. It is not a test of emotional maturity or a personality test, although the responses often reveal important personality traits.

Since the invention of the individual or clinical tests, so-called group tests of intelligence have appeared in great profusion and have attained an enormous vogue as devices for obtaining a preliminary rating, for grouping pupils in sections according to test results, for making a rapid survey of the "intelligence level" of large groups of individuals, or for "screening" pupils for individual examinations.

Merits of the Binet scale as an instrument for classification. This is not the place in which to discuss the virtues of different kinds of tests, verbal or performance and so on, for purposes of classifying intellectual deviates, or the comparative excellences of different tests. A few comments are apropos, however, regarding the merits of the oldest scale, the Binet, which, in spite of its recognized limitations, continues to be the most widely employed instrument for grading children according to general intelligence.

(1) The tests are objective, are given under controlled and standardized conditions, and are supplied with age norms of serviceable, if not complete, accuracy. By means of the scale, intelligence⁴² can be measured much as height or weight is, although less accurately of course. The tests help us to replace personal impression and opinion with an objective measure.

(2) Whether it is absolutely accurate or not, the scale enables us to attach a far more definite connotation to the various terms or categories in our classification of subnormals than was possible before its use. To say that an adolescent is an idiot with a two-year mentality or an imbecile with a four-year mentality by the Binet scale, is to employ common, understandable language, because the words have a perfectly definite meaning. It is notorious that there was formerly

⁴² Intelligence in the sense of the traits and qualities measured by the tests contained in the scale itself, referred to as verbal intelligence by some writers.

little consistency or uniformity in the use of different categories in the classification of mental deficient. The terms idiot and imbecile were applied by different writers to the gravest as well as to the slightest grades of mental defectives. The Binet scale has helped to introduce order in our technical nomenclature.

(3) The Binet classification can be used to supplement and complete the classifications made from various other points of view; in fact, all classifications are mutually supplementary and complementary. Every classification adds something new to our knowledge of an individual. Our knowledge of a microcephalic or a hydrocephalic mental defective is rendered more complete if we know that the former is an imbecile with a four-year Binet age or an I. Q. of 30, and the other a moron with an eight-year Binet age or I. Q. of 50. The Binet rating is particularly valuable in connection with the educational and socio-industrial classifications, which will be discussed in the next chapter.

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Chapter 7

CLASSIFICATION ACCORDING TO EDUCABILITY AND SOCIO-VOCATIONAL COMPETENCY

The two criteria—educability and socio-vocational competency—of developmental maturity or acquired proficiency are treated together because sharp lines of demarcation between them cannot be drawn in practice. Socio-vocational (or socio-occupational or economic) competency depends in part upon educability and can be enhanced by appropriate educational procedures.

CLASSIFICATION ACCORDING TO EDUCABILITY

Present and potential educational proficiency

The major inquiries regarding the child's educational status assume three forms: what is his accomplishment level or stage of advancement in the various curricular branches at the time of the examination? how much educability, or potential capacity for educational advancement, does he possess? to what extent can he be improved in the various literary branches, in the different forms of sensori-motor efficiency, in craftsmanship (occupational and vocational pursuits), in physical development, in the formation of fixed, desirable, and dependable habits of thought, feeling, action, social and moral response, and application, and in the appreciation of ethical, civic, esthetic, and cultural ideals?

All degrees of educability exist from the modicum of idiocy to the vast possibilities of supergenius. From the standpoint of the legal determination of mental defectiveness the

question of surpassing moment is: can the defective child be educated or trained to care for himself physically, mentally, socially, and morally, to support himself, and to lead an independent existence in society?

*Methods of determining attainment level
and educational potentiality*

Four methods have been widely used to determine whether a child is "at age" in his grade placement, or is educationally retarded or accelerated.

The curriculum standard. The ordinary standard of measurement is the ability of the child to meet the requirements of the prescribed curriculum. The validity of this criterion is predicated on the assumption that the difficulty of the subject matter has been accurately adjusted to the educational capabilities of the typical child of the standard age for each grade. Scores of surveys of "pedagogical retardation" during the first two decades of the century (beginning with the investigation of William H. Maxwell in the New York public schools in 1904 and the more extensive investigation by Leonard P. Ayres in 1909), aroused the suspicion that the standards were pitched too high because of the large amount of nonpromotion, failure, retardation, or overageness. Although there has been considerable improvement with the passing years in the grade placement of subject matter and in better adjustment of teaching and learning procedures to the different maturity and ability levels found in the regular grades, this standard of measurement is subject to many limitations. Among these is the fact that children are not always promoted in accordance with sheer educational readiness or achievement. One of the most disturbing practice of automatic promotion in order to keep the child with his own age group.

Objective statistical methods. THE AGE-GRADE METHOD. The child is recorded as "at age," "overage," or "underage" on the basis of the age standard adopted as the normal age for

a given grade. One of the difficulties with this measuring device is the lack of uniformity regarding the normal age standard for each grade. The original standard of 6-0 to 6-11 for the first grade was rapidly abandoned because it often yielded from 50 per cent to 75 per cent of overageness or educational retardation, which was commonly called "pedagogical retardation." To introduce uniformity Heck suggested an age span for 1B grade from 5-9 to 6-9 and for 1A from 6-3 to 7-3, and that the age be calculated from September first. Others allow a span of two years (from 6-0 to 7-11 for the first grade) instead of a year and a half.

Nearly all age-grade studies reveal far more overageness than underageness. Thus in Cincinnati in 1907, 58.4 per cent of the pupils were retarded and only 9.6 per cent were accelerated. The corresponding percentages for three Chicago schools the same year were 68.1 per cent and 8.1 per cent. In 1920, 23.5 per cent of the white pupils in the elementary grades in St. Louis (exclusive of those in special classes) were retarded from one to eight years, whereas only 6.5 per cent were accelerated from one to four years, based on ages six and seven as the standard age for the first grade. The overage pupils were from 8 to 10 times as numerous as the underage ones in 29 cities investigated by Ayres. Thirty-seven cities in 1922-23 yielded a median percentage of retardation of 38.8 as against 10.2 of acceleration.¹

The ratio of educational retardates has probably decreased as a result of better adjustment of the program of education to individual differences and the practice of automatic promotions. Thus in all the Delaware schools for white pupils (exclusive of Wilmington) the number of overage pupils declined from 26.6 per cent in 1931-32 to 16.7 per cent in

¹ For a recent survey and bibliographies of the problem of nonpromotion, retardation, and school failures see the articles by Henry J. Otto (pp. 441 f.) and Merle R. Sumption (pp. 1054 f.) in *Encyclopedia of Educational Research*. New York: The Macmillan Company, 1941. See also Louttit, *Clinical Psychology* (revised edition), pp. 236-256.

1945-46.² Whether the decline is mainly due to earlier entrance or better adaptation of the instruction to individual pupil needs, or mainly to the practice of automatic promotion, which has been growing in some schools, cannot be determined from the available data. Even in the school year 1945-46, the spread of chronological ages for grades I to VIII averaged 6.8 years per grade in the schools for the white (with a larger age range in the schools for the colored).³

THE PROGRESS STANDARD. According to this standard, the educational status and potentiality of the pupil is determined by the amount of time required to perform a unit of work successfully, or the amount of work actually accomplished in a unit of time. A table so constructed might show the percentage of pupils who progressed at the rate of 50, 75, 100, or 125 per cent, or who required two semesters to do the work of one semester, or who covered a year's work in one semester. On the basis of the progress ratio (the number of semesters of promotion to the number of semesters of attendance), the number of grade pupils who had failed one or more semesters was 71.3 per cent in Omaha, 26.7 per cent in 347 consolidated schools in Iowa, and 24.4 per cent in Davenport, Iowa,⁴ during the period of vigorous advocacy of 100 per cent promotions.

COMBINED STANDARD. This is a combination of the age-grade and progress standards and shows the time required by the child to reach a given grade.

VALUE OF METHOD. These mass methods aid in enumerating the number of retarded, on-time, and accelerated pupils and the number making slow, retarded, or accelerated prog-

²And the percentage of underageness from 8.2 to 6.7, based on the normal age standard of 6 to 7-6 for grade I and 7-6 to 8-6 for grade II. See State of Delaware, *Annual Report of the Department of Public Instruction for the Year Ending June 30, 1932*, p. 234; for the Year Ending June 30, 1946, p. 242.

³The 1946 report, p. 242.

⁴Stroud, James B., "How Many Pupils Are Failed?" *The Elementary School Journal*, February, 1947, 316-322.

ress, but they do not tell us why particular children are educationally retarded or advanced or why they deviate to the extent that they do. They do not indicate what proportion of the educationally retarded children are mentally deficient, all-around backward intellectually, educationally retarded but mentally normal, or mentally normal but victims of educational or social neglect, mental inhibitions, emotional or nervous instabilities, or physical handicaps. The data published by Stroud and the numerous earlier studies of grade repetition have shown unmistakably that a large proportion of overage-ness is due to nonpromotion because of failure to meet the grade standards, but not all such failures can be ascribed to inherent lack of intellectual ability or of educational potentials. The exact composition of a motley group of educationally retarded children can only be determined by an individual study of each child. Statistical methods of studying retardation have their place, but they are not clinical and throw little light on the basic causes of educational variations or anomalies.

Standardized composite educational measuring scales. Few accurately standardized clinical (individual) composite educational measuring scales, consisting of tests in a variety of subject matter fields arranged in an ascending order of difficulty in either age-steps or grade-steps, have thus far been devised.⁵ This is strange in view of the obvious utility of such tests in clinical practice. Among the available batteries of tests is the educational attainment scale by Porteus, which consists of tests in reading (word calling), reading comprehension (based on oral presentations and replies to questions), spelling, and numbers.⁶ A convenient system of scoring has been provided, and, to a limited extent, age and grade scores.

The brief Wallin-Gilbert (now Cutsforth) "educational at-

⁵ For a brief description of the early unstandardized scales or batteries of educational tests see Wallin, *Clinical and Abnormal Psychology*, pp. 35-37.

⁶ Porteus, *Studies in Mental Deviations*, pp. 222 f.; *The Practice of Clinical Psychology*, pp. 249-254.

tainment scale for clinical use,"⁷ consists of standardized tests in writing, written language, reading, phonetic spelling, and arithmetic, with grade norms based on 1,066 pupils enrolled in grades one to three in two public schools in Dayton, Ohio. The test can be administered in about ten minutes.

The Jastak "wide range achievement test," with grade norms from the kindergarten to the college level, is limited to the three literary fundamentals: the oral reading of words in columns (word pronunciation), the writing of dictated words given in the order of difficulty, and the solution of computation problems in addition, subtraction, multiplication, and division. The tests have been standardized on the basis of thousands of pupils tested singly and in groups.⁸

Contrasting with the dearth of standardized clinical composite educational scales is the large number of group standardized composite achievement scales or batteries of tests. Among the best known achievement batteries on the elementary level are:

- New Stanford Achievement Tests
- Metropolitan Achievement Tests
- Modern School Achievement Tests
- Public School Achievement Tests
- Progressive Achievement Tests
- Unit Scales of Attainment

These tests, covering grades I or II to upper high school grades, differ somewhat as to content, but most contain tests of reading (word recognition, word, paragraph, or sentence comprehension), spelling, arithmetic fundamentals (computation, problem solving), and possibly capitalization, punctuation, English usage, history, and nature study.

Standardized achievement tests in separate subject matter

⁷ Wallin, J. E. W., and Gilbert, Margery, "A Brief Educational Attainment Scale for Clinical Use," *The Pedagogical Seminary and Journal of Genetic Psychology*, September, 1927, 446-489.

⁸ Jastak, J., *Wide Range Achievement Test*. Wilmington, Del.: Chas. L. Story Co., 1941.

fields. Tests, standardized as to method of administration, scoring, and interpretation, in almost all areas of instruction offered in the elementary and secondary schools and in higher institutions are available in printed booklets conveniently arranged for administration and scoring. Standard scores are supplied in terms of grade norms (and in some cases theoretical age equivalents) and E. Q.'s—educational quotients, obtained by dividing the chronological age into the educational age, the latter often obtained by transmuting the grade score into an age equivalent. Sometimes measures of dispersion are also supplied, such as standard deviations, variances, quartile deviations, percentiles, critical ratios or scores, and so on. When the intelligence age and the educational age are available, the A. Q. (attainment or achievement quotient) can be computed by dividing the intelligence age into the educational age. In theory at least, the A. Q. shows whether the child's educational attainments correspond to his intellectual ability. An A. Q. of 90 shows that the child is accomplishing 10 per cent less than the average child of his intelligence age, whereas an A. Q. of 110 shows that he is accomplishing more than is to be expected, and may be applying himself too assiduously. The A. Q. is offered as a scientific device for comparing any pupil with a standard pupil and for measuring the educational influences of the school, teacher, home, and the child's industry. The A. Q., however, is no more accurate than the premises from which it is computed, the intelligence and educational ages.

A number of so-called diagnostic tests have been devised to reveal the exact deficiencies in certain phases of a given subject matter field as a basis for remedial instruction or for revealing specific proficiencies or aptitudes. For example: What is the precise nature of the child's punctuation difficulties? Do they involve the comma? If so, in which of its many uses? The child may be asked to supply the correct punctuation in mimeographed sentences involving various uses of commas, semicolons, periods, and such. Again, what are the pupil's specific

weaknesses in arithmetic? Is the difficulty an isolated one or only a phase of a more general problem? Does it involve carrying in addition, the addition of fractions, or some phase of percentages?

The Compass Diagnostic Tests in Arithmetic contain over 90 distinct items for determining the strength or deficiency in as many aspects of arithmetic ability. One student found over a hundred chances of errors in solving simple problems dealing with decimals.

In the field of reading, specific weaknesses or disabilities revealed by diagnostic reading tests include reversals (such as *p* for *q*, *b* for *d*, *saw* for *was*, *no* for *on*), line skipping, alteration of vowel sounds (such as *not* for *note*) or consonant sounds (such as *his* for *this*, or *so* for *go*, *ping* for *swing*, *cober* for *cover*), addition of sounds (*swing* for *sing*), omission of sounds (*sing* for *singing*), downright substitution of dissimilar or unrelated words (*by* for *of*), repetition of words (*a girl a girl had a doll a doll*, for *a girl had a doll*), the addition of words (*once upon a time I saw*, for *once I saw*), the omission of words (*a dog*, for *a little dog*), and the like.

Among the best known batteries of diagnostic reading tests are those of Monroe and Durrell,⁹ and two of the most comprehensive pieces of screening apparatus for testing vision are the Keystone Telebinocular¹⁰ and the Massachusetts Vision Test developed by the Massachusetts Department of Public Health.¹¹

Most of the so-called diagnostic educational tests are de-

⁹ Monroe, Marion. *Children Who Cannot Read*. Chicago: University of Chicago Press, 1932. Durrell, Donald D., *Analysis of Reading Difficulty*. Manual of Directions, Yonkers: World Book Company, 1933.

¹⁰ Betts, Emmett A., *Prevention and Correction of Reading Disabilities*. Evanston, Ill.: Row, Peterson and Company, 1936, pp. 161-169, 310-381.

¹¹ The test, made by Welch Allyn, Auburn, N. Y., and approved by the Council of Physical Therapy of the American Medical Association, consists of (1) the Snellen symbol E test, projected on an electrically illuminated screen, for the determination of visual acuity (myopia), (2) a plus sphere test for determining hyperopia (longsightedness), and (3) the Maddox rod test for heterophoria (muscle imbalance). These are screening tests; they cannot be used for determining refractive errors.

scriptively diagnostic rather than causally or etiologically diagnostic. That is, they may reveal the presence or absence of certain specific abilities of which the total ability in a given subject matter field is composed. They may reveal the detailed nature of the child's specific difficulties, say in reading, without at the same time revealing underlying causes, whether they are psychological, social, or physical. Nearly all of the existing tests and scales measure the educational level which the child has already reached as a result of prior instruction and not the potential ability or educability which he may possess in the various branches of study, although, by repeating the tests at various intervals, a measure may be obtained of the amount of progress made, which will furnish an indirect measure of the degree of educability. In the absence of any instruction whatever, few of the existing educational tests give a measure of the child's potential educational capacities.

The prime essential for providing genuine remedial instruction is to determine the exact nature of the child's subject matter difficulties and their etiological background. Although a well-trained, experienced teacher may be able to determine a child's particular needs without the use of standardized tests and plan his work effectively in accordance with his total needs, some of which may not be revealed by tests, any good test properly used will aid the skilled teacher to arrive more quickly at a fuller appreciation of the child's strengths and weaknesses.

Importance of the educational classification

The educational classification, especially the determination of a defective child's degree of educability, is highly important from the practical viewpoint, for two reasons. (1) The problem of the improvement or amelioration of mentally defective and backward children of the primary hereditary type is pre-eminently an educational rather than a medical problem, because of the fact that this type of mental subnormality

is not an active disease nor the result of an active disease process that is subject to cure or improvement by medication or surgical interference. Primary mental deficiency is not a disease but a defect, or a condition of arrested, imperfect, or damaged cerebral development which, although not eradicable, improves with growth and especially as the result of appropriate literary, motor, occupational, social, and hygienic training. This view is shared by Schröder, psychiatrist and one-time director of the University Neurological Clinic in Leipzig, who conceded that "congenital intellectual debility is in most cases as little a disease as is genius. . . . The education of the majority of feeble-minded children is the task of the teacher."¹²

The basic orthogenic process for a considerable number of backward children whose backwardness is constitutional rather than environmental is educational rather than medical. Indeed, much emphasis in the treatment and prevention of all types of functional mental disorders (psychoses) is now placed on re-education, occupational and social diversion, mental prophylaxis, the inculcation of right mental attitudes toward the problems of life, the establishment of healthy habits of thought, feeling, and response, the proper hygiene of the emotions and organic drives—in a word, on "mental hygiene."

(2) It is highly important to know whether the defective child can be educated to assume a normal independent position in life, or whether he must occupy a dependent position and require control, supervision, or custody. This knowledge is of more vital importance than the knowledge that the child is a microcephalic, or macrocephalic, or that the cause is environmental or hereditary. Information of the latter type is highly interesting but it is of secondary importance for the practical handling of any particular mental defective.

¹² Patry, Frederick L., "A Résumé of Reports and Discussions in 'School Psychiatry,'" *Journal of Exceptional Children*, January, 1939, 88.

Limitations of the educational classification

In spite of its importance, the educational classification is subject to a number of limitations. (1) Educability depends on many factors other than innate "strength of mind," "inherent capacity," alertness, or intelligence. The other factors include interest, enthusiasm, ambition, effort, application, learning readiness, learning attitude, early training, age on entering school, regularity of attendance, the cultural advantages afforded by the habitat, memory (ordinary school tests and many standardized educational tests place a premium on memory rather than on judgment or understanding), emotional drive, emotional blocking or instability, health, and specific physical, mental, or educational defects. Therefore, a child's educational deficiency may be due to any one of these adventitious factors or a combination of them, rather than to the inherent lack of ability.

(2) Educability depends not only on the condition or capacity of the learner, but also on the skill and personality make-up of the teacher. Some failures in school have made normal progress when transferred to another teacher. The justification for lengthening the teacher training program is the belief that the well-trained teacher achieves better results in pupil training and growth than the inadequately prepared teacher.

(3) The degree of educability, as determined by the school standards or the subject matter taught in school, is not always a reliable criterion for making predictions regarding the child's ability to succeed in life. Some children who have failed miserably in literary learning have achieved decided success in many practical pursuits.

(4) Children cannot be classified educationally with accuracy until their degree of educability has been determined by actual trial, and this takes time. Sometimes a very considerable interval may elapse before the teacher is able to reach a positive decision. What is needed are reliable means

for determining educability beforehand; that is, whether the child has the "general intelligence" required for success in certain subject matter fields, and the specific potentialities needed for success in reading, spelling, arithmetic, and other branches. Tests of general intelligence are available in profusion, as we have already seen, from the results of which the child's general intelligence potentiality can be predicated. But the construction of tests that show whether the child possesses the specific potentialities needed to achieve success in different branches of instruction remains a challenging problem, although the existent "readiness tests" and "aptitude tests" have some value along this line. The purpose of the writer's phonetic spelling scale, based exclusively on phonetic words, was, in part, to supply a measure of potential spelling capacity, but not enough use has ever been made of the scale to justify an opinion of its value from this point of view.¹³

Moreover, the investigations of the last few decades have shown rather conclusively that the child's educability cannot be determined reliably in the absence of a favorable learning attitude or emotional undertone—a liking for the subject matter and for the teacher, emotional security, and self-trust and enthusiasm engendered by the joy and satisfaction of successful achievement. Innate potentialities may remain dormant or may be inhibited by unfavorable mind sets produced by emotional conflicts, repressions, fears, anxieties, and hostilities. It may require a long time of morale development to remove the effects of emotional inhibitions and to free latent capacities for constructive achievement. Witmer discovered that Don, who had been diagnosed as a low grade mental defective, was really a case of pseudo-feeble-mindedness. Morbid fears of new persons or things had induced excessive inattention and a condition simulating feeble-mindedness. When the fears were dissipated, the child's development proceeded with surprising rapidity.

¹³ Wallin, J. E. W., and Coles, Charlesanna B., "Phonetic Spelling Scales." *The Pedagogical Seminary and Journal of Genetic Psychology*, 1928, 431-470.

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Martin Barr's educational classification

Our immediate concern is the question how mentally subnormal children can be classified from the standpoint of educability. One of the earliest and most complete classifications is Barr's. Barr divides subnormals into four groups, with rather elaborate subgroupings. The best of the lowest grades, "idio-imbeciles," can only be trained to help themselves and to assist others to a limited extent and require "asylum care" for life. The highest grades of the "moral imbeciles" are "trainable in manual and intellectual arts," but have a "genius for evil" and require "custodial life and perpetual guardianship." The high grade "imbeciles" possess similar educational possibilities, but require "long apprenticeship and colony life under protection," and the "backward and mentally feeble" are "trainable for a place in the world."¹⁴

The Vineland and Waverley classifications are to a considerable extent educational classifications, as occupational proficiency is based in large measure on educability or trainability.

CLASSIFICATION ACCORDING TO SOCIO-VOCATIONAL
COMPETENCY

*Meaning of social, economic, occupational,
or vocational capacity*

Children differ greatly in the rate of acquisition of various personal, social, and vocational competencies. Information regarding the child's level of personal, social, and occupational proficiency can be obtained by the methods of observation, interview, and controlled tests.

¹⁴ For details see Barr, M. W., *Mental Defectives*. Philadelphia: P. Blakiston's Son & Co., 1910, p. 90. Other early educational classifications are those of Weigand and Felix Voisin. Barr's high grade imbeciles obviously correspond to the morons in the Vineland Industrial Classification.

Where social and occupational proficiency can be observed

In the *home* can be observed the child's aptness to gain control over his powers and to acquire habits of self-help—to sit, walk, talk, control the bladder and bowel reflexes, feed himself with spoon, fork, and knife, wash himself, dress and undress, button and unbutton—and to conform to the rules and regulations of the home, to run errands, and to perform the ordinary chores and tasks in and about the house or farm. Every parent should make and record observations on such developmental facts as these and compare the results with the developmental norms that are available in the writings on child growth. Systematic records of actual observations should serve as a corrective of the slipshod appraisals that many parents supply to the case worker and of the errors of memory that often serve as a defense against facing disagreeable facts.¹⁵ Some parents are notoriously blind to defects in their own children, particularly if the children are mild-mannered and well-behaved. A mother who brought a feeble-minded child from Illinois reported that she never suspected anything was wrong with him until she received a school report after his second year in the first grade. She had noticed nothing abnormal in his conduct at home, but a neighbor with whom I conferred saw that the child was "not right" at the age of three or four. It is surprising how frequently the question, "When did you first discover there was anything wrong with the child?" will evoke the reply from the mother, "not until he entered school." The tendency of some parents to blind themselves to defects in their own progeny is sometimes due to unrecognized distortions of memory that serve as "escape mechanisms."

On the *playground* children are unconsciously sized up by their fellows and peers, and unconsciously classify themselves by the way they seize the opportunities afforded to display

¹⁵ For methods of improving observational studies see Olson, Willard C., *The Measurements of Nervous Habits in Normal Children*. Minneapolis: University of Minnesota Press, 1929.

initiative, leadership, courage, physical prowess, strength, quickness, intelligence, judgment, resourcefulness, honesty, fairness, and control of the emotions and disposition. The playground affords excellent opportunities for observing children's ability to cooperate, understand and execute directions, plan, originate, lead, obey, conform to the rules of the game, subordinate individual interests, forget self in the interest of group success, and marshal all forces for the attainment of a definite goal in the possible face of odds. One of the great advantages of observing the playground activities is the opportunity afforded to study the child when he is interested, absorbed, dynamic, spontaneously active—that is, as G. Stanley Hall has remarked, when the child is most truly himself. Play is the universal language of childhood.

In recent years psychologists and psychiatrists have turned to the observation of children's spontaneous undirected play activities as a means of therapy and of diagnosis of problems and maladjustments. Children often express their inner fantasies, personal problems, frustrations, anxieties, longings, motivations, and interests in their free play activities. The child's imaginative plays often symbolize his inner struggles and cravings. He is usually the suffering or conquering hero in such play activities. His plays often afford outlets for his inner tensions. The hostility toward her mother or teacher may be expressed, perhaps, in the spankings a girl administers to her doll.

The playground estimates of capacity are valuable and often accurate, but far from infallible. By no means do all of those who display dominant personalities on the playground later ascend to positions of prominence in the trades or professions.

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Finally job performance in the schools, fields, factories, shops, and offices affords splendid opportunities for observing children's character traits. In the job it is possible to note special interests and talents, habits of industry, vocational ability, ability to understand instructions, follow directions, analyze situations, and plan solutions, and the power to acquire new motor habits and skills.

The method of the interview

A vital supplement to the method of observation is the interview, whether in the free or standardized form. The free, informal interview is the traditional device used by coun-

selors, advisers, guidance workers, and social workers for obtaining an over-all picture of the client—his response patterns, his problems, frustrations, difficulties, interests, aspirations, attitudes, alibis, defenses, maladjustments—for establishing rapport and friendly relations; and for imparting information, encouragement, and suggestions. The method has more recently been employed to point the conversation to the client's problem without direct questioning so as to lead him to work out his own solution, and to give vent to his feelings and express himself without restriction and without direct suggestions, directives, or prescriptions from the therapist in an encouraging, accepting, permissive atmosphere (nondirective and release therapy). Interview techniques are more flexible than test procedures and serve useful purposes, but they are less accurate and more subjective and impressionistic.

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Types of tests and scales for determining social, industrial, occupational, and vocational competencies

Test procedures are designed to yield a more scientifically precise and accurate evaluation of the client's mental resources: his general ability level, specific abilities or disabilities, his aptitudes, dormant talents, trained or developed skills, detailed personality characteristics, and the like. It would bring us entirely too far afield to discuss all the devices that have been used in years past to determine socio-occupational proficiency, vocational interests and skills, personal and social adjustments, or almost any component of the personality. Many of the techniques used have only an indirect value for social or occupational prediction or for vocational guidance, preparation, and placement. This synopsis is limited to an enumeration of various tests and other devices used under the major groupings and a brief discussion of some of them.

(1) Educational or attainment tests in almost all subject matter fields, single and in batteries, group and clinical, for the determination of achievement level, placement, discovery of specific abilities or disabilities (clinical function), and for the determination of capacity to acquire or inherent aptitude (predictive or prognostic function). Illustrations of tests of this nature were given in the preceding section.

(2) Verbal tests or scales of general intellectual ability or mental alertness, clinical and group. The clinical tests include some versions of the Binet, the Wechsler-Bellevue (partly performance), the Detroit Test of Learning Aptitude, and the new Army Individual Test. Among the numerous

group tests of general intelligence may be mentioned various versions of the Army Alpha, the Henmon-Nelson Tests of Mental Ability, the Otis Quick Scoring and Self-administering Tests of Mental Ability, the California Tests of Intelligence or Mental Maturity, the Illinois General Intelligence Scale, the National Intelligence Tests, the Pintner General Ability Tests, Verbal Series, the Dearborn Group Tests of Intelligence, and the Chicago Tests of Primary Mental Abilities.

General ability tests of this nature, whether group or individual, are not highly prognostic vocationally¹⁶ and do not give much insight into specific talents, but they possess great value for determining the general level of intellectual ability and the relative brightness of the examinee. It is important to have this determination at least for those who deviate conspicuously in either the minus or plus direction.

(3) Clinical and group tests of general motor or psychomotor ability, performance level, or general mechanical aptitude. The different tests include scales or batteries of various kinds of form boards or manipulative materials, such as the Arthur Performance Scales, the Pintner-Paterson Scale of Performance Tests, the Cornell-Coxe Performance Ability Scale, the Goodenough "Drawing-a-Man" Test, the Porteus Maze Test, the Stenquist Mechanical Assembly Tests, and the new Oseretsky Tests—a year scale of tests of motor maturation from age four through age sixteen arranged on the Binet pattern.¹⁷

Among the best known batteries of performance tests for younger children are the Merrill-Palmer Scale of Tests, the

¹⁶ On this point, see the comments in Wallin, *The Education of Handicapped Children*, and the references given to the experimental literature, pp. 362 f., 378. Also Pintner, R., *Intelligence Testing*, pp. 471 f.

¹⁷ De Costa, Maria I. L., "The Oseretsky Tests," *Training School Bulletin*, March, 1946, 1-13; April, 1946, 27-38; May, 1946, 50-59; and June, 1946, 62-74 (translated by Elizabeth J. Fosa). For annotated references consult Lassner, Rudolf, "Annotated Bibliography on the Oseretsky Tests of Motor Proficiency," *Journal of Consulting Psychology*, January-February, 1948, 37-47.

Minnesota Preschool Scale, the Gesell Developmental Schedules for Infants, the Bühler Baby Tests, and the Psyche Cattell Scale for Infants and Young Children.

The group tests, or paper-and-pencil tests, include the Army Beta, the Pintner-Cunningham Primary Mental Test, the Pintner General Ability Tests, Nonlanguage Series, the Stenquist Mechanical Aptitude Tests, the Detroit General Aptitudes Examination, the Detroit Mechanical Aptitudes Examination, the Minnesota Paper Form Board Test, the O'Rourke Mechanical Aptitude Test, and the MacQuarrie Test for Mechanical Ability. The latter, perhaps the most widely used of general mechanical ability tests, consists of seven subtests that measure the accuracy of tracing, speed of tapping dots, speed and accuracy of dotting, the perception of space relations, and the accuracy of eye movements.

Psychomotor or mechanical aptitude tests correlate better with practical motor ability than do the verbal tests. They serve a very useful function in connection with tests of verbal intelligence in the diagnosis of mental defectiveness. Many who test mentally defective by the verbal intelligence tests display so much ability in the performance tests as to justify a suspension of judgment of defectiveness in practical manipulative skill.

(4) Group and individual tests, verbal, pictorial, and performance of specific aptitudes, or for the discovery of the presence of the specific potentialities needed for the development of proficiencies in many occupations or vocations (mechanical prediction or prognosis tests). Among such tests are those designed to determine whether the learner possesses the traits or the talents needed to become a successful typist, stenographer, teacher, musician, physician, engineer, aviator, pilot, truck driver, and the like. Many of these tests are highly specific, such as tests of finger dexterity, precision of movement, fineness of motor coordination, speed of reaction, the ability to distinguish small differences in pitch, or to reproduce pitch intervals. For instance, in one of the best known

tests of speed of movement, the Minnesota Rate of Manipulation Test, the subject replaces 60 round blocks in round recesses as fast as possible and then turns each block over and replaces it as rapidly as possible. This test is stripped of unnecessary complications to reduce the test to one of speed.

Fortunately, many children of low general ability possess sufficient specific aptitudes along some line to enable them to become economic and social assets.

The greatest spurt in the development of aptitude test batteries has come since Pearl Harbor through the work of the War Department and the war industries. Hundreds of jobs and vocations have been analyzed to determine the abilities and personality qualifications essential for success in different jobs. Few of the tests have thus far been released by the army or by industry. Aptitude batteries now exist for about 170 occupations. Moreover, many jobs and occupations have been broken down into job families. Breakdowns into about 85 families of related jobs and occupations are now available in employment offices for use in connection with transferring and in upgrading workers. These occupations and breakdowns are based on analyses of job and worker's traits—dexterity, muscular strength, fineness of motor coordination, memory, and other needed traits. Some experimenters entertain the hope that it may be possible to subdivide all occupations into ten or fifteen basic families based on the presence of the same primary traits as shown by factor analysis. Should such reductions or combinations prove feasible, the problem of occupational testing would be greatly simplified. This job has not yet been accomplished.¹⁸

One of the most significant advances in the future in the field of vocational counseling and placement will, probably, be the development of aptitude tests designed to reveal the presence or absence of the basic mental capacities needed for success in many trades and professions.

¹⁸ Wallin, J. E. W., "The Psychological Aspects of the Problem of Vocational Preparation and Rehabilitation of Mentally and Physically Handicapped Civilians," *American Journal of Mental Deficiency*, January, 1945, 298.

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The program of analysis of trades and professions (sometimes referred to as vocational analysis), of the specialties within trades ("occupational analysis"), of particular operations on a particular piece of material on a particular machine ("job analysis and job specification"), and of personnel specifications did not begin, however, with World War II. During the first world war "trade specifications" were formulated by the Division of Personnel in the army for 714 army civilian trades and occupations.¹⁹ These specifications showed the detailed duties attached to each occupation, the qualifications required by the workers, and the kinds of occupational experience or training which could be substituted for the recommended specific training or experience.

All these significant developments should prove of value in the training and placement of at least the higher grades of mentally defective and subnormal adolescents.

(5) Verbal, pictorial, and performance tests of acquired trade skills. In connection with the program of job and occupational analysis and trade specifications, the psychologists

¹⁹ *The Personnel System of the United States Army*. War Department Document, I and II, 1919; *Trade Specifications and Index of Professions and Trades in the Army*. War Department Document No. 774.

With respect to World War II consult *Personnel Classification Tests*. War Department Technical Manual 12-260, Washington: Government Printing Office, 1946; Stuit, Dewey B., *Personnel Research and Test Development in the Bureau of Naval Personnel*. Princeton, N. J.: Princeton University Press, 1947.

in World War I before mobilization closed constructed and standardized the "army trade tests" for 83 of the more essential trades in the army. By means of these tests it was possible to determine whether the job applicants for appointment already possessed trained skill for different jobs and occupations and, if so, the degree of skill possessed.

The *verbal* tests were based on oral or written answers to oral or written questions regarding technical details of the trade in question. The *pictorial* tests were based on oral answers to oral questions concerning a given trade situation depicted in pictures, the naming of the tools, machines, or parts of machinery shown, and designation of their uses. This form of test was devised to overcome language difficulties or to serve as a check on coaching. The *performance* tests were based on the actual execution of a typical job, requiring the use of the principal tools, from a blueprint or printed directions. Many additional tests of this nature have been devised since World War I by the psychologists employed by industry and government departments. The value of these measuring devices in the field of scientific placement of normal as well as subnormal workers is obvious.

(6) Interest inventories, questionnaires, tests, checklists, and attitude scales, applying to many phases of life, including vocational preferences.²⁰ The assumption in this kind of appraisal is that the employee will function best in the field of his interests and that a significant correlation exists between interest and aptitude or capacity. Although the latter assumption is commonly justified, many exceptions exist. Thus Maggie, a fourteen-year-old subnormal girl with a Binet level of about eight years, had the ambition to become a teacher. It was very difficult to persuade her that her limited talents lay in some other field.

²⁰ For a discussion of these and personality tests see Symonds, Percival M., *Diagnosing Personality and Conduct*. New York: D. Appleton-Century Company, 1931; and Greene, E. B., *Measurement of Human Behavior*.

(7) Personality tests in great profusion—such as tests of introversion, extroversion, ascendance, dominance, aggressiveness, submission, fears, worries, self-reliance, emotional instability, emotional immaturity, mood, submerged complexes, neurotic make-up, racial prejudices, social skills, etc.²¹ The value of these tests for the more exact measurement of personality intangibles has not been fully established as yet, particularly so far as concerns children of limited intellectual endowment, but they present an interesting challenge to the psychological frontiersmen of the decades ahead.

(8) The Vineland industrial classification. This classification of mental defectives (Table III)²² lists the representative tasks which typical mentally defective children under twenty were able to do in each Binet age in the Training School at Vineland, according to the reports submitted by the attendants, teachers, and employees. Binet ages ten to twelve are omitted because the results are based on few observations and these levels of ability as determined by the 1908 Binet scale do not necessarily predicate mental defectiveness in the social sense.

It should be realized that this classification is based upon a limited number of juvenile institutional cases and the results should not be applied uncritically to institutional adults or to noninstitutional subnormal or normal children of the same Binet age levels.

The 1947 revision of the somewhat similar but far more detailed Scale of Industrial Possibilities, constructed at the Walter E. Fernald School in Massachusetts, shows the minimum Binet "ages at which a large number of children are able to do any given kind of work." The schedules of learning possibilities in occupational pursuits from Binet ages three to ten

²¹ See Symonds, *Diagnosing Personality and Conduct*, pp. 41-121, 171-259, 361-399, 450-457, 503-535; also Porteus, *The Practice of Clinical Psychology*, pp. 164-197 (references); Thorpe, Louis P., *Psychological Foundations of Personality*. New York: McGraw-Hill Book Company, Inc., 1938, pp. 519-585.

²² *Journal of Psycho-Asthenics*, XV, September-December, 1910, 62, 134.

are shown in Table IV.²³ The schedules do not, of course, reflect all the possibilities of every child of a given Binet level.

TABLE III
Vineland Industrial Classification

Binet-Simon Age		Classification	Capacities
Under 1	Low	Idiots	(a) Helpless. (b) Can walk. (c) Capable of voluntary regard.
1	Middle		Feeds himself but eats everything.
2	High		Eats discriminately.
3	Low	Imbeciles	Plays a little but does no work.
4			Tries to help.
5	Middle		Does only the simplest tasks.
6	High		Does tasks of short duration.
7			Washes dishes.
8			Does little errands in the house.
9	Morons	Morons	Dusts.
			Does errands and light work. Makes beds.
			Does heavier work. Scrubs. Mends.
			Lays bricks. Cares for bathroom.

²³ Raymond, C. Stanley, "Industrial Possibilities of the Feeble-Minded Within an Institution," *Proceedings of the Fifth Annual Session of the American Association for the Study of the Feeble-Minded*, June, 1926 (reprint). Appreciation is expressed to Malcolm J. Farrell, M. D., Superintendent, for the privilege of publication of the scales.

For a tabulation of suitable occupations, based on the studies of several investigators of noninstitutional deficient, intelligence ages five to ten, see Louttit, *Clinical Psychology*, pp. 226-227. Attention should also be called to the Porteus Scales based on the study of institutional defectives at the Vineland Training School: the Social Rating Scale, based on the relation of personality defects to social maladaptation or inadequacy; and the Industrial Rating Scale, based on a job analysis of needlework, woodwork, shoe repairing, broom-making, and other occupations. See "A Study of Personality of Defectives with a Social Rating Scale," No. 23. Vineland, N. J.: The Training School at Vineland, 1920; "A Social Rating Scale for Defectives," *Journal of Psycho-Asthenics*, XXVI, 1924, 117-126; *Studies in Mental Deviations*.

TABLE IV

*The Walter E. Fernald State School Schedule of
Industrial Training for Imbecile and Moron Boys of School Age*

Three-Year Mental Level

- Pick up balls scattered by teacher.
- Bead stringing.
- Peg boards, large pegs.
- Learn to hold hammer and to pound hammer or mallet.
- Learn to lace shoes.
- Use of button strips.
- Carry stones and sticks from one pile to another in play yard.

Four-Year Mental Level

- Bead stringing by form and color.
- Coloring with crayon.
- Oilcloth weaving mats.
- Peg boards, small pegs.
- Use of hammer:
 - Hold hammer correctly (Use waste lumber and 10d. nails).
 - Pounding in rhythm (class exercise).
 - Pounding nails at random.
- Simple sandpapering of flat surface.
- Learn to thread needle.
- Learn to take off and put on outer clothing.
- Tie shoe laces.
- Picking up paper and stones.

Five-Year Mental Level

- Bead stringing by color, number and form.
- Use of hammer:
 - Pounding nails to line.
 - Pounding nails to intersection of lines.
- Use of saw:
 - Holding saw correctly.
 - Sawing at random.
- Thread needle and knot thread.
- Paper weaving mats (School).
- Begin Todd loom weaving.
- Begin braid loom weaving.
- Learn to use scissors.
- Spool knitting.
- Begin brush making.
- Simple cleaning tasks in shop and on wards.

Six-Year Mental Level

- Use of saw:
 - Sawing to line.
 - Begin use of coping saw.
- Sandpapering.

Brush making (scrub, flesh, radiator, tampico brushes).
Practice painting—applying flat coat.
Cutting coir yarn to equal lengths.
Making coir yarn mats.
Braid weaving; table mats.
Todd loom weaving.

Seven-Year Mental Level

Carpentry—rough work; learning use of tools.
Sandpapering.
Cane seating, practice work using heavy twine and frame.
Painting—flat work.
Coir yarn mats.
Coir braid mats.
Begin rug weaving.
Todd loom weaving with pattern.
Braid weaving; rugs.
Brass craft; punched book-ends, blotter corners, etc.
Leather work; book marks, belts.
Dishwashing, bedmaking, setting tables.

Eight-Year Mental Level

Painting—flat work, lawn furniture, beds, coat hangers, etc.
Cane seating.
Weaving—rug and towel looms.
Broom making (sorting corn, sewing).
Painting—flat work, toys.
Carpentry—coat hangers, toys.
Weeding and harvesting vegetables.
Helping to serve food and waiting on table.

Nine-Year Level

Weaving—good work on rug and towel looms.
Shoe repairing—rough nailing, ripping.
Broom making; whole process.
Carpentry—boxes, bookcases and racks, footstools, end tables.
Painting—fine work.
Furniture and bed repairing, under close supervision.
Operating foot power printing press.
Blocking paper in pads.
Pillow making.
Hair cutting—shampooing, shaving with safety razor.
Market gardening under supervision.
Harvesting vegetables and preparing them for use at kitchen.

Ten-Year Level

Printing; setting and sorting type, care of type and press.
Shellacking, varnishing, staining.
Sign painting and other skilled painting.
Mattress making, under supervision.
Carpentry; fine work.
Glass setting.

Furniture and bed repairing; less supervision required.
 Shoe repairing; whole process.
 Gardening as above; less supervision required.

*The Walter E. Fernald State School Schedule of
 Industrial Training for Imbecile and Moron Girls of School Age*

Three-Year Mental Level

Pick up papers and balls scattered by teacher.
 Bead stringing.
 Peg boards, large pegs.
 Lacing stick; learn to lace shoes.
 Learn to button and unbutton, using button strips.

Four-Year Mental Level

Bead stringing by form and color.
 Coloring.
 Learn to thread needle.
 Learn to weave over and under on oilcloth mat.
 Roll bandages.
 Lacing and tying shoes.
 Take off and put on outer clothing.

Five-Year Mental Level

Bead stringing by color, number, and form.
 Thread needle and knot thread.
 Begin plain sewing with large needle, colored thread; running stitch on scrim of coarse material.
 Sew rags for rugs.
 Begin Todd loom weaving.
 Begin braid loom weaving.
 Learn to hold and use scissors.
 Spool knitting.
 Simple cleaning tasks in shop and on wards.

Six-Year Mental Level

Sewing; sampler with variety of stitches.
 Outline stitch, for toys, rag dolls, holders, etc.
 Cut pictures for scrapbooks.
 Crocheting—chain stitch; begin.
 Knitting—simple stitch; begin.
 Todd loom weaving with pattern.
 Rug weaving, hit or miss pattern (older girls).
 Net bag making—laundry bags, basketball baskets, tennis nets (older girls).
 Begin rug hooking.
 Work in paring room (older girls).
 Bed making, setting tables, cleaning tasks on wards.

Seven-Year Level

Rug weaving, with pattern.
 Scarf weaving on table loom.
 Cutting and pasting pictures for scrapbooks.

Crocheting face cloths and other articles requiring single crochet.
 Knitting—plain stitch.
 Cross stitch, outline stitch, lazy daisy embroidery.
 Simple hooked rug patterns begun.
 Begin domestic science work.
 Dishwashing, waiting on tables in dormitory.

Eight-Year Mental Level

Embroidery—more advanced work.
 Weaving; rug, towel, napkin material.
 Scarf loom: 4-harness table loom.
 Crocheting; scarfs, caps, etc.
 Knitting; knit and purl—scarfs, caps, etc.
 Learn to run sewing machine.
 Plain sewing; doll clothes and other plain work.
 First steps in basketry.
 Bead loom weaving.
 Brass work: punched book-ends, etc.
 Hooked rug making under supervision.
 All types of housework on wards.

Nine-Year Mental Level

Embroidery—wall hangings, table covers, curtains.
 Knitting socks, mittens, sweaters.
 Crocheting fancy edgings, doilies, etc.
 Operation of hand knitting machine to make stockings and socks.
 Basketry.
 Toy making; stuffed toys.
 Operation of jigsaw to make picture puzzles.
 Running household power sewing machine.
 Making of slips, dresses, and other garments cut by more advanced pupils.
 Mending of stockings and garments.
 Tatting.
 Rug hooking; complete process with good results.
 Pillow lace making.
 Operation of 4-harness loom.
 Plain cooking.
 All types of laundry work (older girls only).

Ten-Year Mental Level

Knitting and crocheting table covers, bedspreads, altar cloths, sweaters, etc.
 Leather work; pocketbooks, book covers.
 Embroidery—complicated patterns.
 Artificial flowers.
 Running commercial power sewing machine under supervision.
 Dressmaking; cutting, fitting, and making garments, under supervision.
 Plain cooking.
 Planning and serving meals, under supervision.
 Scrapbook making.
 Assisting teacher in bookbinding process. Laundry work continued.
 Beauty parlor operator: shampooing, hair cutting, marcel and finger waving.
 giving of home permanents under supervision.

The Vineland Social Maturity Scale. This is not a scale of tests but of reported observations by parents or others of various everyday abilities of a child, such as grasping objects within reach, sitting unsupported, walking without assistance, drinking from a cup or glass, eating with a spoon, communicating in short sentences, playing with children, removing the coat or dress, washing the face unassisted, making telephone calls, and the like. The scale consists of 117 items arranged in age steps from 0.1 to 25 in "an approximate order of increasing social significance" or "growth in social responsibility." Each test measures "some particular aspect of the ability to look after one's self" in social situations. A few tests evaluate the child's ability to participate in social activities "which lead toward ultimate independence as adults."²⁴ The results of the tests are stated in terms of S. A. (social age) and S. Q. (social quotient).

The relative extent to which ratings obtained by the scale are dependent upon environmental opportunities (paucity or richness of cultural milieu) and upon inherent social potentialities (or native social endowment) and the relative value of S. Q.'s obtained from this scale and I. Q.'s obtained from the Binet scale for the diagnosis of mental defectiveness are questions that cannot at present be answered with finality.

The suggestion has come from some investigations that the S. A. and S. Q. give a better evaluation of the defective's practical ability than the Binet age and I. Q. and that they thus supply a more reliable tool for the diagnosis of mental defectiveness in its socio-legal connotation. Thus Doll remarks: "We have definite indications that this scale does provide a valuable means of distinguishing between the definitely feeble-minded who are socially inadequate and the intel-

²⁴ Doll, E. A., *The Vineland Social Maturity Scale: Revised Condensed Manual of Directions*, No. 3. Vineland, N. J.: The Training School at Vineland, April, 1936; "Annotated Bibliography on the Vineland Social Maturity Scale," *Journal of Consulting Psychology*, July-August, 1940, 123-132; *Vineland Social Maturity Scale, Manual of Directions*. Minneapolis: Educational Test Bureau, 1947.

lectually subnormal who are socially adequate.”²⁵ Be that as it may, the scale adds another battery to the armory of the child examiner. With all the aids at his command, the expert on mental defectiveness should be able to free his psychological diagnoses from the errors that were characteristic of an earlier day.

Importance of the socio-vocational classification

The accurate appraisal of socio-vocational capacity, actual or potential, is of paramount importance from the viewpoint of recommendations regarding the kind of training to be provided and the ultimate disposition to be made of a deficient child. Ultimately we must face the question whether the child has sufficient capacity or acquired competency, general or special, to earn his living in some useful pursuit, and whether he has sufficient social ability and stability to exercise self-control, to manage his own affairs with reasonable discretion, to assume an independent position in society, to assume social obligations, and to conform to the laws and conventions of society; or whether he is so defective intellectually, emotionally, volitionally, and socially, and incompetent industrially, that he will always remain dependent and more or less irresponsible and possibly potentially or actually delinquent. We cannot wisely plan a handicapped child's course of training without attempting some sort of answer to these questions. The larger problem in the case of the mentally defective is social: the proper protection of society from the consequences of mental defect—whether pauperism, criminality, or biological contamination through the reproduction of the biologically or socially unfit—and the protection of the helpless and dependent from neglect, starvation, and from being imposed upon and duped by the predatory wolves of

²⁵ “Preliminary Standardization of the Vineland Social Maturity Scale,” *Journal of Orthopsychiatry*, April, 1936, 293; Doll, Edgar A., and McKay, B. Elizabeth, “The Social Competence of Special Class Children,” *Journal of Educational Research*, October, 1937, 90-105.

society. The problem includes the protection of the irresponsible from the consequences of their folly or evil propensities; and the salvaging of all the assets possessed by defectives for productive, constructive work under custody, supervision, parole, or the liberties of citizenship. The basic criteria for the diagnosis of feeble-mindedness, on which the legal definition is framed, are psychological and sociological.

Chapter 8

CLASSIFICATION ACCORDING TO ETIOLOGY

Classification according to cause

According to the nature of the cause (technically known as *etiology* in the field of medicine), mentally deficient children may be classified in two ways: first, according to the nature of the factors or circumstances that directly or indirectly produced the defect (called *pathogenesis*, the science of the origin of a morbid state); and, second, according to the nature of the underlying abnormal structural changes, particularly those in the nerve cells (called *pathology*).

NATURE OF THE CAUSATIVE FACTORS (PATHOGENIC FACTORS)

The causative factors may be grouped into three categories: hereditary, environmental, and mixed (heredo-environmental). Many writers use other terms in preference to the words hereditary and environmental. Among the terms often used as synonyms for hereditary are primary (suggested by Tredgold in 1908), innate, intrinsic, inborn, endogenous, native, constitutional, genetous, and germinal. The terms that are equivalent to environmental are secondary, extrinsic, exogenous, acquired, and somatic. In the primary or inherited form, due to defective (or *cacogenic*) heredity, the cause has operated prior to conception. The mental deficiency is based on a defect or taint in the genes, ultramicroscopic physiochemical elements in the germ plasm. August Weismann applied the latter term to the substance which carries the factors (*i.e.*, genes) of particular hereditary qualities or tenden-

cies and which is transmitted in direct continuity from generation to generation.

Hereditary Factors

In connection with the theory of hereditary transmission, it is imperative to distinguish sharply two kinds of cells: (1) the somatic or ultimate body cells (constituting the tissue of all the bodily organs), which are nontransmissible, and (2) the germ cells, whose chromosomal genes are the bearers of hereditary tendencies. Hereditary defects are, therefore, referred to as germinal.

It is also necessary to draw a distinction between the two concepts, congenital and hereditary. Congenital is the more inclusive term. It means merely that a quality or condition is inborn or that it exists before birth, as a consequence of either hereditary forces or environmental (intrauterine) impacts. A congenital defect may, therefore, be caused by a germinal defect or by a somatic defect produced by the maternal environment. Congenital syphilis, for example, is caused by actual invasion of the embryo or fetus from the mother's organism of the germs of syphilis (*spirochaeta pallida*)—thus it is a somatic defect, and is not caused by infected germ cells. Although it is inborn, congenital syphilis is secondary rather than primary, environmental rather than hereditary.

According to the theory of mechanical evolution, all biological traits and variations of organisms from generation to generation are mechanically determined by the combination of genes from which the individual develops and cannot be altered by his own experiences. Mental defectiveness is merely one form of mechanically predetermined germinal variation from the normal type. According to the theory of "emergent" evolution, on the other hand, the successive steps in evolution result in the production of new forms or variations that cannot be predicted with accuracy from the antecedents, instead of being merely recombinations or resultants

of pre-existing factors. A third group of geneticists, particularly those dealing with psychosocial phenomena, stresses the view that human traits or behavior characteristics are the results of the reciprocal action of heredity and environment. Heredity and environment are not isolated, independent factors, but inseparable, interacting phases of the organism's process of inner and outer adjustment. In other words, trait formation is the joint result of the organism's processes of natural maturation (from cellular differentiation and organic and functional growth, or maturing as the result of intrinsic or hereditary forces) and of the individual's experiences (such as exercise, practice, habit formation, thinking, and learning). Each forward advance in maturation lays the basis for the acquisition of new and more complex reaction patterns.

It is, in point of fact, very difficult in practice to determine whether a defect, congenital or otherwise, is primary or secondary, or the result of the interaction of both factors. This may not be true of some physical characteristics, such as eye or skin color, but it is true of most mental characteristics and deviations. Though all hereditary mental defects may be potentially present at birth, the fact is that many of them cannot be recognized when the child is born. It requires years before some defects become apparent to the parents or even to the family physician. The psychoclinician is frequently told by parents that they did not realize that the child was mentally deficient or backward until he entered school and unfavorable reports on his school progress were received from the primary teacher. My own investigations of the family histories of many thousands of examinees in psycho-educational clinics tend to support the opinion of Penrose that 90 per cent of mental defectives appear normal at birth,¹

¹ Penrose, L. S., *Mental Defect*, p. 166. On the other hand, Gesell believes that a baby's intelligence can be diagnosed with accuracy at birth or within four or five months by means of a standardized behavior and developmental examination and the observation of physical stigmata. Gesell, A. L., "Differential Diagnosis of Mental Deficiency in Infancy," *Nebraska State Medical Journal*, 1947, 23: 304-307.

and also the converse fact that many who appear abnormal at birth eventually prove to be normal. Even a head that is grossly misshapen at birth may right itself without readily discernible evidence of mental impairment. In general, then, primary mental deficiency ordinarily becomes apparent only very gradually.

When the mental deficiency originates after birth, the child shows normal or fair mental development until some trauma (accident) or disease occurs. Then arrest or deterioration supervenes, but this may occur very insidiously, so that it is sometimes difficult to trace it to its exact origin. However, the fact that the mental impairment does not become manifest until some crisis, accident, disease, or developmental strain occurs, does not mean that the brain cells were originally sound and stable. It is generally conceded that nervous systems burdened with neuropathic heredity are less resistant and more susceptible to the ravages of extraneous nerve-destructive forces than are those of normal hereditary potentials. Nevertheless, many neuropathic or potentially unstable nervous systems would have adjusted in a fairly integrated and satisfactory manner to the social and physical situational demands, had it not been for some untoward accident or developmental crisis which precipitated the expression of their hereditary defect.

The modus operandi of hereditary transmission of defects. Discrepant views are entertained regarding the exact mode of transmission of an inherited mental defect and how the abnormal transmission differs from the normal pattern of inheritance. Some have attributed mental defectiveness to an innate, spontaneous process of variation, or to a natural process of gene mutation. Others attribute it to artificially produced gene mutations. Some interpret it as a reversion or an atavistic phenomenon, the appearance of traits that resemble those of earlier or more primitive evolutionary forms. Some construe it as a developmental arrest of hereditary potentials. Some (a negligible number) explain the defect in terms of the Galtonian principles of transmission, and others follow

the Mendelian hypothesis, at least so far as certain types of defectives are concerned.

Thus Davenport advanced the hypothesis in 1912 that mental deficiency represents a defect of the germ plasm (hereditary material) that has "surely come all the way down from man's ape-like ancestors, through two hundred generations or more. The germ plasm that we are tracing remains relatively simple; it has never gained, or only temporarily at most, the one or the many characteristics whose absence we call (quite inadequately) 'defects.' Feeble-mindedness is thus an uninterrupted transmission from our animal ancestry. It is not a reversion; it is direct inheritance."²

Goddard, in emphasizing that "feeble-mindedness is hereditary and transmitted as surely as any other character,"³ reaches the following conclusion from an analysis of extensive genealogical tables: "It is clear from the data . . . that feeble-mindedness is hereditary in a large percentage of cases, and that it is transmitted in accordance with the Mendelian formula."⁴ Goddard, Davenport, Holmes,⁵ and many others have explained mental normality as a Mendelian dominant and mental defectiveness as a simple Mendelian recessive, and have held that the mechanism of hereditary transmission accords with the law of inheritance formulated in 1865 (but forgotten or ignored by scientists until about 1900) by the Austrian abbot who became a distinguished botanist, Gregor M. Mendel (1822-1884). On the other hand, the Committee of the American Neurological Association, after an exhaustive survey of the writings, reached the conclusion "that if there is a Mendelian inheritance, it is multifactorial."⁶ In

² Davenport, Charles B., "The Origin and Control of Mental Defectiveness," *Popular Science Monthly*, 1912, 80: 87-90.

³ Goddard, Henry H., *The Kallikak Family*. New York: The Macmillan Company, 1912, p. 117.

⁴ Goddard, H. H., *Feeble-Mindedness, Its Causes and Consequences*. New York: The Macmillan Company, 1914 (and 1920), pp. 560-561.

⁵ Holmes, Samuel J., *Trend of the Race*. New York: Harcourt, Brace & Co., Inc., 1921.

⁶ Myerson, et al., *Eugenical Sterilization*, pp. 130 f. (the committee finds most studies of the inheritance of mental deficiency inadequate and inconclusive, pp. 113-136).

harmony with this view, Burlingame concludes that "mental deficiency is due in most cases to defective genotypes" (the total inherited constitution of an individual, including all hereditary traits, whether manifest or latent). "Defective genotypes arise from the combinations of multiple cumulative genes."⁷

At the other extreme is the view of Lewis, that the majority of mental defectives merely represent the lower extreme, or the fag end, of the normal curve of distribution of intelligence.⁸ They are the result of the normal biological tendency toward variation from the mode and not the result of pathological processes or neuropathic inheritance. He applies the term "subcultural" to this kind of mental deficiency, which is not the product of defective heredity or pathological environmental agencies. The word "aclinical" has been employed more recently by Halperin in the same sense.

Tredgold, perhaps the leading living medical authority on mental defectiveness, apparently accepts Davenport's theory of a simple, unevolved germ plasm as the basis for mental dullness, but rejects this explanation of mental defectiveness. Mental defectiveness is caused by a general "impairment of the germ developmental potentiality," which might be "consequent upon some nutritive disturbance of the germ cell itself," or "senescence of the cell," or "damage inflicted upon the cell by harmful influences of its environment." The "neuropathic diathesis" (constitutional predisposition), evidenced by the presence of various forms of mental abnormality in the ancestry, is transmitted not as "specific gene mutation, or combination of such mutations, but it is a general impairment of the vitality or developmental potentiality of those genes which are responsible for the growth of the brain."⁹ No appreciable mental abnormality would result

⁷ Burlingame, Leonas L., *Heredity and Social Problems*. New York: McGraw-Hill Book Company, Inc., 1940, p. 282.

⁸ Lewis, Edmund O., "Types of Mental Deficiency and Their Social Significance," *Journal of Mental Science*, 1933, 298-305.

⁹ Tredgold, A. F., *Textbook of Mental Deficiency* (6th edition). Baltimore: William Wood & Company, 1937, pp. 33 f.

from a slight impairment under favorable environmental conditions, whereas a very serious impairment might produce mental deficiency in spite of a favorable environment.

According to the orthodox, Weismannian point of view, "neuropathic inheritance," so-called, represents some kind of intrinsic blight in the hereditary germinal factor in the chromosomes (microscopic bodies in the nucleus of the cell), which has been transmitted solely from the inherently defective genes transmitted from preceding generations, and not from the defective, somatic cells of the ancestors. For example, the child is mentally deficient because his immediate or remote and direct or collateral ancestors were carriers of inherently defective germ plasm. The mental defective in generation number 200, then, is defective because of the neuropathically tainted genes of his forbears in generation 199; the defective in generation 199 is, *pari passu*, defective because of the defective genes in his ancestors in generation 198, and so on *ad infinitum*, without any break through by exogenous forces. Feeble-mindedness, insanity, and epilepsy, it is alleged, constitute the great triad of neuropathic inheritance; and the presence of these abnormalities in the ancestral strains, direct or collateral, constitutes positive proof to many geneticists that the given case is of hereditary origin. This is the view of the doctrine of polymorphism, namely, that any neuropathic condition or form of degeneration may be the cause of feeble-mindedness, mental disease, or epilepsy. This view is completely rejected by the Committee on Sterilization of the American Neurological Association, which holds that there is no essential relationship between these three nervous defects.¹⁰

Since the Mendelian theory of inheritance has been extensively applied as a causal explanation of the simple type and some of the special types of mental defectiveness, it is important to review, in briefest outline, the most important features of Mendelism and comment upon its adequacy as an explanatory hypothesis of this type of defectiveness.

¹⁰ Myerson, *et al.*, *Eugenical Sterilization*, pp. 82-89.

The Mendelian law of heredity and mental defectiveness. Mendel's formulation was based on extensive crossbreeding (or hybridization) of 22 pure varieties of edible peas. He traced the crossing of single pairs of contrasting characters or characteristics, such as round and wrinkled peas, or tall and dwarf peas, during a period of eight years, and found that the characters appeared in the offspring of successive generations in definite, predictable ratios. For example: when pure varieties of smoothly round and wrinkled peas were crossbred or hybridized (the fusion of a male sperm and a female egg from two distinct species or varieties), the resulting offspring (hybrid) in the first filial generation (F1) were all round, like the round-seeded parent. Thus, in the F1 generation the round peas prevailed over the wrinkled ones. When one character or trait suppresses, overrides, or prevents the appearance of the contrasting character (called the recessive), it is referred to as a dominant character. If it is present in one or both parents, a dominant character will appear in the offspring to the exclusion of the contrasting trait in the other parent. A character contributed by both parents (double dominance) is referred to as duplex. It is called simplex when it stems from only one parent, and nulliplex when it is absent in both parents. On the other hand, the planting of the hybrid round peas from F1 yielded in the second generation (F2) round and wrinkled peas in the ratio of three round and one wrinkled peas. Obviously, the F1 round peas were not all pure round because 25 per cent of the offspring were wrinkled. Apparently the wrinkled character had remained latent in the first generation (F1). Such a latent character was referred to by Mendel as a recessive. A recessive may be defined as a character from one parent which remains undeveloped in the progeny when it is associated with the corresponding dominant in the other parent. The recessive appears only when it is not paired and overcome by the corresponding dominant gene, that is, when both members of a pair of genes are alike. Although it does not appear in the

immediate offspring, it may be transmitted to later generations because the character (wrinkled peas) was present in the F₁ generation of hybrids but did not express itself on account of the presence of the dominant (round peas). F₂ gave three kinds of peas: a pure round (duplex), which bred true generation after generation; a pure wrinkled, which continued to breed true because it did not contain the dominant round character (hence was nulliplex with respect to that determiner); and two round peas (simplex) which contained the wrinkled characteristic as a recessive. In succeeding generations the round seeds produced by the hybrid round peas continued to appear in the ratio of one pure bred to two hybrid round peas. Thus, in 30 generations of round peas, one-third produced only round ones whereas the other two-thirds produced round and wrinkled peas.

Every other pair of contrasting hybrids gave three kinds of progeny in approximately the same ratio. The recessives, when self-fertilized, produced only pure recessives; the recessives always bred true.

According to this theory, hereditary characters or traits are transmitted from parents to offspring as independent units from each parent without undergoing any change or exerting any influence upon one another. One parent contributes one unit (*e.g.*, roundness in peas) and the other another unit (*e.g.*, wrinkling in peas). The determiners separate when they produce sperms and eggs. Instead of blending in the process of fertilization, there is segregation. In pure bred varieties the determiners of the characters are "identical," "concordant," "duplex," or "homozygous" (*homo*, same; *zygote*, egg). In hybrids, the sperm and the egg differ with respect to the given determiner and the character is "simplex" or "heterozygous" (*hetero*, different).

The unit of any particular hereditary quality or character (also referred to as a "unit character," "determiner," or "factor") is the gene, the infinitesimally small unit in the nucleus of the germ or reproductive cell (gamete), about one ten-mil-

lionth of an inch long. It has been estimated that each chromosome contains several thousand genes. The exact chemical composition or nature of the gene has not been determined. It is probably a nucleoprotein molecule. The genes are arranged in definite linear order in pairs opposite each other in the chromosomes, which are deeply staining (*chroma*, color) rod-shaped bodies (*soma*, body) in the nucleus of the cell.¹¹ The chromosomes are visible under the microscope at the time of cell division. The number of chromosomes is constant for any species. The human sperm (male) and egg cells (female) each contain 24 chromosomes. Although the ordinary cells (making up the muscles, brain, skin, and other body parts) contain 48 chromosomes, the reproductive cells (the sperm and egg) have only 24 (referred to as "reduction" or meiosis of chromosomes), giving 48 in all in the fertilized egg. Since both parents contribute an equal number of chromosomes to the progeny, they share equally in the child's heredity. Males have one X-chromosome and a smaller mated Y-chromosome, and females possess two X-chromosomes. These chromosomes have to do with sex, in contrast with all other chromosomes, which are referred to as autosomes. When one of the chromosomes is lacking, the egg produces a male instead of a female. The male lacks one chromosome that is present in the female.

According to the traditional view, the ultimate physical units of heredity, the genes, influence the body (somatic) cells but are not influenced by them. Heritable traits may be due to one or more dominant genes, to one or more recessives, to ordinary or sex-linked recessives, or to some combination of these. The genes act separately and independently. The more recent view is that the genes are not simple, fixed, static, independent units or entities that are transmitted according to a simple mathematical formula, but dynamic, complex combinations of diversified chemicals that react upon one

¹¹ Thomas H. Morgan seems to have been the first person to localize the hereditary process in the cell nucleus of the chromosome.

another in a complicated way, as do chemicals in a test tube.

Each parent possesses thousands of genes, usually arranged in pairs. Each pair probably contains related chemicals. The father contributes one gene from each of his pairs and the mother contributes the corresponding gene. A single hereditary character represents a synthesis of genes, not the result of the action of one gene. Thus, the red eye color in the fruit fly is the result of the interaction of at least 20 genes.¹² The absence of a particular gene is not the absence of a particular character but of a chemical agent. The absence of a single gene will affect the result somewhat. Each gene may affect the organism differently according to the combination of genes.

No two individuals are ever alike with respect to their gene composition except identical twins, which originate from a single fertilized egg. Each person is genetically unique. Mathematically the probability that two siblings (offspring of the same parents) would possess a combination of identical chromosomes is "one to hundreds of trillions."¹³

There are "several lines of thousands of genes in each human cell"¹⁴ and any inherited characteristic is the result of the combined action and interaction of many genes. A given gene may affect many traits and interact in the production of a single trait. Moreover, the characteristics that any individual develops are not due solely to his genes, but to the joint action of the genes and the environment. Each individual's uniqueness is due to both gene differences and environmental differences. The genes cannot develop in a vacuum; they can function only in cooperation with indispensable environmental conditions. Both gene potentials and light are necessary for the development of the green color in plants, for example. Tanning from ultra-violet light does

¹² Altenburg, Edgar, *Genetics*. New York: Henry Holt & Company, Inc., 1945, p. 15.

¹³ Osborn, Frederick, *Preface to Eugenics*. New York: Harper & Brothers, 1940, p. 4.

¹⁴ *Ibid.*, p. 1.

not occur in the absence of the pigment potential in the gene, as is the case with albinos. A variety of Chinese primrose produces red flowers at 20 degrees C, but white flowers at 30° C or above. Both colors can be obtained from a single plant by moving it from one temperature to another. The fur of the Himalayan rabbits becomes white when the temperature is warm, but dark when it is cool. Dwarfs can be produced in plants and animals by withholding certain essential vitamins and proteins. Traits develop anew each generation under the joint influence of heredity and environment.

Very slow variations in terms of geologic time may occur in the genes as a result of exceedingly slow fortuitous variations around the mean and as a result of the natural selection of the more favorable variants produced by such environmental changes in food, temperature, moisture, light, altitude, geographical isolation, sexual selection, and so on that may make for the "survival of the fittest" (Charles Darwin). More rapid changes may be brought about by crossbreeding, chance distortions, and mutations (Hugo DeVries, 1901). Mutations are sudden gene changes that result in new species, often called sports, which are preserved by natural selection if the changes are favorable to the animal's survival. They represent permanent, transmissible variations. Mutations and recessives in general prove to be injurious and do not make for the improved adaptation of the organism. The mutated forms sometimes return to the normal type by a process of reverse mutation.

A half century of experimental research has apparently demonstrated that many physical traits in plants and animals, and perhaps some specific types of mental defect, are transmitted in accordance with the Mendelian law or some modification of it. Corresponding proof, however, in support of the hypothesis that the simple primary type of mental deficiency is inherited as a Mendelian recessive has not been

supplied. The evidence accumulated by Goddard¹⁵ is based on pedigree charts which extend many generations into the shrouded past and obviously do not admit of verification with respect to the diagnosis made of mental defectiveness or of its genetic basis. The Mendelian explanation of the simple type of mental defectiveness has not yet been demonstrated to be correct from the standpoint of acceptable standards of genetic research. Neuer's pronouncement in a recent article is even more explicit: "The assumption that a heredo-degenerative nervous disease or personality disorder might be caused by a single dominant or recessive factor is untenable in the light of modern research."¹⁶ In a later study of the etiology and clinical diagnosis of 300 institutional cases of "mental deficiency" he affirms categorically that "faulty heredity" is "only a minor cause" even of the simple, nonorganic type which apparently, on the basis merely of recorded data (none too accurate) in the personal and family histories, he attributes to "emotional disturbances, inferior cultural milieu, and substandard economics."¹⁷ In point of fact, he classifies only 1.7 per cent of all his cases as hereditary, one of the lowest estimates of hereditary transmission of mental defectiveness on record. Of his cases, 32.4 per cent are classified as "simple" cases, all others being grouped as "organic" (39.3 per cent) and "neuropsychiatric" (28.3 per cent). Of the 87 neuropsychiatric cases (a vague, omnibus category), 39 were diagnosed as neurotics and 21 as psychotics, exclusive of 7 manic-depressives and 5 postencephalitic psychotics (why these were not included among the psychotics is not clear), and 15 were undifferentiated. These unusual results require critical evaluation.

One psychological implication of the Mendelian hypothesis

¹⁵ Goddard, *Feeble-Mindedness, Its Causes and Consequences*.

¹⁶ Neuer, H., "Prevention of Mental Deficiency," April, 1947, 724.

¹⁷ Neuer, H., "The Relationship Between Behavior Disorders in Children and the Syndrome of Mental Deficiency," *American Journal of Mental Deficiency*, October, 1947, 143-147.

which militates against its acceptance as an explanation of the simple or aclinical type of mental defectiveness is discussed in Chapter 3. This, however, would not prove that the cases might not be of genetic origin.

Mental deficiency from gene injury (blastophthoria). For a century or more a voluminous amount of studies on observations of human matings, animal and plant experimentation, theorizing, positive claims and equally emphatic counter-claims from the same data have appeared in the scientific press on the question of the causation of mental and physical defects by the modification or degeneration of the gonadal (reproductive) cells, and particularly the genes, from parental toxication by such poisons as alcohol, nicotine, lead, mercury, arsenic, iodine, alkaloids, opium, morphine, and ether.¹⁸ The investigations of the present century have included the effects of the injection of antibodies into the blood stream, maternal rubella, the Rh blood factor, mustard gas, the chemical colchicine, varying the temperature, light, color, and moisture changes in the environment, deprivation of oxygen and essential food elements, the production of acidosis, repeated amputations, ultra-violet, ultrasonic, atomic, X-ray, neutron, or radium radiation, uranium nitrate, and the like. The radiation experiments, with the direct application of the rays

¹⁸ For a discussion of the genetic effects of alleged "racial poisons," see Carroll, Robert S., *What Price Alcohol*. New York: The Macmillan Company, 1941, pp. 34 ff.; Frets, Gerrit P., *Alcohol and Other Germ Poisons*. The Hague: Martinus Nijhoff, 1931. Meggendorfer, Friedrich, in volume 3 of "Die Erbliche Fallsucht der Erbveitanz und der Schwere Alcoholismus," of Arthur Gutt's *Handbuch der Erbkrankheiten*, Leipzig, 1940, pp. 382 f. Also Wallin, *The Education of Handicapped Children*, especially pp. 291-300.

C. R. Stockard's apparently conclusive experiment on the transmission of hereditary defects from parental alcoholization of guinea pigs through many generations, was discredited by the nine-year investigation along similar lines undertaken by the British Medical Research Council. Paul Kammerer's exhaustive experiments on gene mutations produced by moisture and color changes in the external environment were discredited by the "doctoring" of an assistant who, unknown to Kammerer, produced the changes artificially by means of aniline dyes. The discovery of the cruel hoax caused Kammerer to commit suicide. For an evaluation of some of the material, see Myerson, *et al.*, *Eugenical Sterilization*, pp. 69 f.

to the reproductive cells, have been carried out on a great variety of animals and plants.

The scope of this book does not permit a summary of the extensive experimental investigations on gene mutations in animals or of the studies of the progeny of alcoholized and nicotinized human parents and of the conflicting results and claims of the different investigators. However, a few observations are apropos.

Briefly, the essential facts seem to be about as follows. By means of the umbilical cord the embryo is directly connected with the mother's placenta. But no direct connection exists between the nervous and blood systems of the mother and the embryo. The blood vessels merely lie side by side and the embryo obtains its nutriment from its mother's blood by a process of absorption or diffusion. No reason exists for assuming that only the nutritive elements of the mother's blood are absorbed by the child's blood vessels and that antibodies and the toxins of disease and poisons (especially alcohol and nicotine) are effectively screened out. The absorption of toxins from maternal toxemias does not come to a complete halt at the portal of the placenta. When the pregnant mother is alcoholized or nicotinized, the embryo shares in the intoxication. Experiments have shown that the rate of the heart beat of the fetus is speeded up when the mother smokes. The average increase in one experiment was five beats a minute. Unborn babies smoke when their mothers smoke, and go on a "binge" when their mothers are "on a jag." The drug sulfanilamide administered to pregnant women in therapeutic doses (for the treatment of gonorrhea or pyelitis) passes into the fetal blood within five hours in nearly the same concentration as in the maternal blood,¹⁹ apparently with possibilities of injury to mothers and children who are allergic to

¹⁹ Heckel, George D., "Chemotherapy During Pregnancy: Danger of Fetal Injury from Sulfanilamide and Its Derivatives," *Journal of the American Medical Association*, October 18, 1941, 1314-1316.

this drug. Although toxication of the embryo and the fetus may, and doubtless does, produce injury to the somatic cells, it has not been demonstrated conclusively thus far that the damage is reflected in the genes. Some geneticists go so far as to say that the genes lead an immutable, sacrosanct existence, completely protected against external violence. Although the genes are doubtless highly protected against harmful agencies by the nuclear membranes which surround them—some poisons probably destroy the living cells before they can penetrate the layer of cytoplasm and reach the gene at the nucleus—yet experiments have shown that the spermatozoa can be modified by acids and that the genes and chromosomes in plants and animals can be modified by low and high temperatures, by the chemical colchicine, by mustard gas, and particularly by the X-rays and similar waves, which are more penetrating than many poisons, and that gene changes thus produced may continue as permanent mutants and be transmitted to posterity.²⁰

The accepted conclusions that new species can be developed from a gradual accumulation and transmission of slight fortuitous gene variations which make for better adaptation to the environment (the survival of the fittest by natural selection) and that permanent gene mutations can be artificially produced by environmental agencies (such as radioactive substances and apparently certain chemicals also) suggest that many other substances may exist that might produce destructive hereditary (gene) alterations under certain circumstances and also that slight gene deteriorations might accumulate throughout the generations until they eventually produce cacogenic or dysgenic effects on the progeny, manifested by increased susceptibility toward the development of nervous or psychosomatic disturbances. The experiment of

²⁰ H. J. Muller is acclaimed as the first to demonstrate experimentally (in 1927 on the fruit fly *Drosophila*) that permanent artificial gene mutations could be produced by Roentgen rays. Muller, "Artificial Transmutation of the Gene," *Science*, July 22, 1927, 84-87.

Bluhm at least suggests the possibility of gene variations from alcoholization that has continued for many generations. His experiments on white rats continued for five or six hundred generations. Physical mutations were found to occur after several hundred generations had been subjected to alcohol.²¹ With the wide lacunae that still exist in our knowledge of human genetics and gene variations, aberrations, and mutations, the better part of valor is to avoid the tendency toward premature dogmatization and retain an open mind on the questions of the modifiability of the genes by destructive agencies and the transmission to the progeny of modifications thus produced.²²

The proportion of mental defectiveness that can be attributed to defective inheritance under any of the hypotheses mentioned above (spontaneous variation, natural or artificial mutation, Mendelian recessives, reversion, and so on) will be considered elsewhere. It is now known that certain types formerly ascribed to defective heredity are environmentally produced (cases produced by birth asphyxia and pelvic X-ray-ing). This knowledge will result in the prevention of the birth of a limited number of mental defectives. Great progress has, however, not been achieved thus far in the elimination of hereditarily defective stocks. The current methods of colonization during the child-bearing period, sterilization, and birth control reach only some of the more obvious cases and almost none of the less easily identifiable and more numerous carriers (recessives) who, although not feeble-minded, are allegedly the product of tainted germ plasm and, therefore, capable of transmitting the defect when mated with persons having similar gene defects.

²¹ Quoted from Landis and Bolles, *Textbook of Abnormal Psychology*, p. 266.

²² The euthenic implications of the poisoning of the body cells of the person who absorbs alcohol, opium, nicotine, lead, and other poisons is a question quite apart from the eugenic aspects. The organic and functional disturbances, transient and permanent, produced by many poisons are well known.

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Environmental Factors

Acquired forms of mental deficiency. The secondary or environmental causes include any agencies which injure the cerebrum (brain) through direct action upon the somatic cells after the union of the sperm and the egg. The brain damage may be done before, during, or after birth. The causative factors may thus operate antenatally (through the maternal organism), natively, or postnatally (through the external physical and social environment). The extent of the cerebral lesions (hurts, injuries, or degeneration of parts) and the subsequent mental impairment will vary markedly with the extent of the brain hemorrhage, inflammation, or mechanical injury, with the location of the cerebral damage.

with the nature of associated complications, with the child's age, and with the native resistance of the neurones. The effects of the unfavorable environmental factors may be superimposed upon a neuropathic background, an enfeebled nervous system, and thus intensify the mental defect, or the external factors may injure or destroy nerve cells that were originally normal. Some of the secondary causes produce more devastating mental effects than do the primary factors; and the brain lesions produced by inflammatory processes cause more mental deterioration than those produced by traumas; but many exceptions to this rule occur. In general, the younger the child, the more serious will be the mental defect. The cerebral deterioration produced by some external causes, for instance, hemorrhages, remains stationary after the disease has run its course, and the child will improve gradually as a result of further maturation and training. On the other hand, the mental deterioration is progressive in some cases, for example, in some cases of encephalitis or untreated juvenile syphilitic amentia, and may continue until the child becomes a low grade ament or dement.

The exact time limit of the postnatal period beyond which the mental arrest or deterioration would represent a process of dementia (degeneration subsequent to complete maturation) rather than of amentia (the English equivalent for mental defectiveness; literally, without mind or nondevelopment of mind) has not been determined and probably cannot be determined with any high degree of precision. Some would limit the period to the first few years of childhood; others would extend it into the period of adolescence.

Classification of secondary causes. A multitude of environmental factors have been suggested as possible causes of mental deficiency and inferiority, some of which are responsible for some of the special clinical or physical types. The most important may be listed as follows, with brief evaluations based on the writer's clinical experience.

ANTENATAL FACTORS. Serious maternal diseases of an in-

fectious nature during gestation, such as typhus, typhoid fever, German measles (rubella) during the first two months of pregnancy,²³ malaria, cholera, or syphilis: all these sources probably constitute a relatively small number.

Toxins in the maternal blood stream from lead poisoning (plumbism), nicotine, poisonous drugs, especially alcohol, or injury to the embryo from the unsuccessful use of poisonous abortifacients; these toxic agencies may, in the aggregate, be of considerable importance, especially alcoholic indulgence during gravidity.

Serious functional disorders in the ductless or endocrine glands (called endocrinopathies), particularly in the mother and in the fetus: of undoubted influence in certain types and possibly of more general importance than has been recognized in the past.

Serious maternal nutritional deficiency productive of serious fetal malnutrition: perhaps of greater importance than thus far recognized.

The relation of an inadequate maternal diet to reduction in the weight, length, vitality, and rate of ossification of the bones in the baby, and to increased difficulties of birth and to maternal diseases and toxemias of pregnancy has been shown in a number of studies.²⁴ A marked diminution in the weight and length of the newborn of starved women in western Holland was noted in 1945. Later, upon the improvement of maternal nutrition, the fetal weights returned to normal. In animal experiments the formation of the fetal skeleton has been inhibited by withholding riboflavin (B_2) from the maternal diet, and congenital eye defects have been produced by vitamin A deficiency.²⁵ A maternal rachitogenic diet (yellow corn meal 76, gluten 20, and calcium carbonate) in rats has resulted in delayed calcification and

²³ Consult the references in Shock, Nathan W., "Growth and Development," *Review of Educational Research*, December, 1947, 17: 362, 368-370.

²⁴ For a summary of studies and references see *ibid.*, pp. 336, 368-370.

²⁵ Dispensa, Johnette, and Hornbeck, Richard T., "Can Intelligence Be Improved by Prenatal Endocrine Therapy," *Journal of Psychology*, 1941, 12: 209-224; "Can Intelligence Be Improved by Endocrine Therapy before Fertilization," *ibid.*, October, 1942, 235-243.

bone and cartilage malformations in many of the offspring.²⁶ The defects can be prevented by supplying the needed food elements in the right proportions in the maternal diet—iron, phosphorus, calcium, dried liver, riboflavin, or vitamins A, B complex, C, and D.

The extent to which mental deficiency or retardation, transient or permanent, can be produced by maternal dietary deficiency is in need of more extensive and accurate determination.

The placental passing of antigens (substances that produce antibodies, which are defenses against disease, when introduced into the blood) from the mother into the fetus, shown in incompatibility in the Rh blood factor (discovered by Karl Landsteiner and Alexander S. Wiener in 1940 and named from the initial letters of the rhesus monkey on whom the experiments were carried out) in the mother and in the child: this is thought to cause an indeterminate number of cases of mental defectiveness.

The Rh may be negative in the mother and positive in the child. The Rh antigen is, it is held, transmitted according to Mendelian principles through a pair of autosomal genes (non-sex chromosomes). The dominant is designated Rh and the recessive rh. The child is a homozygous (pure) Rh negative when the rh factor is inherited from both parents; and an Rh positive when the Rh factor is inherited from both parents or when the Rh factor comes from one parent and the rh from the other. In the latter case the child is heterozygous.

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²⁶ The following are some references to the numerous dietary experiments on rats by Joseph Warkany and his associates: *Science*, 1940, 92: 383; *American Journal of Diseases of Children*, 1942, 64: 860-866; *Archives of Pathology*, 1942, 34: 375-384; *American Journal of Diseases of Children*, 1943, 66: 511-516; *Journal of Nutrition*, 1944, 27: 477-484.

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Yannet, Herman, and Lieberman, Rose, "The Rh Factor in the Etiology of Mental Deficiency," *American Journal of Mental Deficiency*, October, 1944, 133-137; "Central Nervous System Complications Associated with Kernicterus," *Journal of the American Medical Association*, February 9, 1946, 130:335-340; "Further Studies on ABO Isoimmunization, Sector Status and Mental Deficiency," *American Journal of Mental Deficiency*, 1948, 314-317.

Direct injury inflicted on the embryo or fetus from violent blows, or falls, or unsuccessful attempts at abortion by means of instruments: these factors will account at most for a small proportion of cases.

NATAL FACTORS. Primogeniture (state of being the first-born) may be a contributory factor, particularly in nervously unstable stocks, because of the greater likelihood of difficult

births and cerebral hemorrhages or other birth injuries among the firstborn. However, Dayton, on the basis of extensive investigations, reached the conclusion that abnormal labor and first births are not linked with mental defect, although the relation between abnormal labor and first births was unquestionable.²⁷ The allegedly greater incidence of mental deficiency among the lastborn is perhaps of questionable significance,²⁸ except in special types, such as mongolism.

Prolonged or difficult parturition, producing asphyxia (suspended breathing from suffocation or oxygen deficiency in the blood), congestion of the brain membranes enveloping the brain (meninges), rupture of superficial blood vessels, and intracranial hemorrhages; anomalies of presentation (twisting of the cord, breech), prematurity or precipitate delivery; improper use of anesthetics; brain injuries by means of forceps; prolonged labor and difficult delivery resulting in asphyxia may be more important than was formerly thought to be the case.

The existence of serious cerebral birth injuries can be recognized from the symptoms in a large proportion of the cases at birth or within a few weeks or months although some signs do not appear for years. The symptoms include marked paleness or cyanosis (blueness of the skin);²⁹ feeble-

²⁷ Dayton, N. A., "Abnormal Labor as an Etiological Factor in Mental Deficiency and Other Associated Conditions: Analysis of 20,473 Cases," *New England Journal of Medicine*, August 28, 1930, 398-413; "Order of Birth of Mental Defectives," *Journal of Heredity*, May, 1929, 219-224.

²⁸ Penrose, *Mental Defect*, pp. 66 f. On the question of the relation of the mother's age at the child's birth to the child's mental capacity, see also Dayton, Neil A., and Truden, Bernardine, "Age of Mother at Birth of Child and Incidence of Mental Retardation in the Children: Study of 23,422 Families of Public School Children Examined by Fifteen Massachusetts Traveling Psychiatric School Clinics, 1921-1935," *American Journal of Mental Deficiency*, 1940, 190-200; Thurstone, Louis L., and Jenkins, Richard L., *Order of Birth, Parent Age, and Intelligence*. Chicago: University of Chicago Press, 1931; Tredgold, *Textbook of Mental Deficiency* (6th edition), pp. 41, 210.

"Blue asphyxia" occurs in about 90 per cent of the newborn according to Frank R. Ford and is usually regarded as trivial compared with "pallid asphyxia" which points to vasomotor collapse from a damaged medulla. Frank L. McPhail and Earl L. Hall classify babies as severely asphyxiated (1) in whom breathing and crying are delayed for more than one minute; and (2) all who develop cyanosis during the first few days of life.

ness and irregularity of the pulse and respiration; bulging of the anterior fontanelle (soft, unossified spot on top of the head); great restlessness, often followed by listlessness, abnormal quietness, somnolence, stupor, or torpor; crying in a high-pitched voice; inability to swallow; disturbances of various reflexes—of the face, eyes, hands, wrists, legs, toes; stiffening of the body or muscular rigidity, retraction of the head, muscular twitchings (possibly from bleeding over the cortex), or general convulsions within a few days which may be followed by motor paralysis (often spastic); awkwardness; failure to sit up; perceptual disturbances, failure to notice surroundings, lack of interest, and retarded mental development. Not all of these signs are present in all cases and not all appear at the same time. In point of fact, damage may be done to the brain without any neurological signs in early life, as shown by follow-up investigations many years later. The symptoms that develop in any given case will depend upon the part of the brain that is injured. Any part of the brain may be involved as well as the lower centers (basal ganglia), the so-called extrapyramidal system. The latter is an accumulation of nervous tissue about six inches below the cortex, consisting of the corpora striata, the thalamus (or interbrain), and other parts. The thalamus (probably an organ of adjustment for cutaneous, visual, auditory, and emotional impulses) is particularly vulnerable to brain injury. Of 127 cases of brain injury, 76 involved the brain and 61 the basal ganglia.³⁰

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Hern, K. M., *Physical Treatment of Injuries of the Brain and*

³⁰ Strauss and Lehtinen, *Psychopathology and Education of the Brain-Injured Child*, citing A. Wald.

Allied Mental Disorders. Baltimore: The Williams & Wilkins Company, 1947.

Strauss, Alfred A., and Lehtinen, Laura E., *Psychopathology and Education of the Brain-Injured Child.*

The researches of the last decade have shown the vital importance of adequate and uninterrupted oxygenation of the brain. Oxygen is the most vital element required by the brain cells. Although the brain constitutes but one-fortieth part of the body, it consumes 10 per cent of the body's oxygen output, and the gray matter of the cortex (consisting of the cell bodies) requires ten times as much as the white matter (the nerve fibers).³¹ The neurones are extraordinarily sensitive to the lack of oxygen. Oxygen deprivation (known as anoxia and apnea) during a very limited period of time may produce devastating cerebral injury. The "survival time of nervous tissue without oxygen is limited to 20 seconds," according to Kugelmass. According to Yant,³² complete lack of oxygen for one minute may have fatal consequences, and Schreiber states that oxygen lack for a "few minutes can produce irreparable damage."

The causes of the interference with the circulation of the oxygen supply are numerous: for instance, strangulation; anemia or reduction of the red blood cells; lack of biochemical balance between the blood constituents (for example, lack of dextrose, which is needed for normal oxidation); low blood pressure, or impaired heart action; injury to the respiratory center in the medulla (the center of cerebral oxidation); poisoning of the blood from carbon monoxide, paraldehyde, narcotic drugs, nitrous oxide (used in connection with dental extractions), sulfanilamide, and various anesthetics and analgesics. In the fetus, anoxia may be produced by

³¹ According to Sherman, "in the developing brain the white matter is more susceptible to oxygen deprivation whereas in the adult brain the cortex is more vulnerable." Sherman, Mandel, *Intelligence and Its Deviations*. New York: The Ronald Press Company, 1945, p. 136.

³² Yant, William P., *et al.*, *Studies in Asphyxia*. Public Health Bulletin 211, Washington: Government Printing Office, 1934, p. 211.

oxygen deficiency in the mother, excessive pressure on the fetal head, difficult birth, and prolonged labor, with associated asphyxia and intracranial hemorrhage (which further respiratory failure), prematurity (the most important cause of neonatal asphyxia, according to Cole and Kimball,³³ based on 5,000 maternal cases), and the ingestion by the mother beyond the limits of safety of drugs to produce "twilight sleep" and to lessen the pains of childbirth (such as morphine, the barbiturates, scopolamine) or to retard labor until the doctor arrives (such as nitrous oxide, ether, or chloroform). These drugs can reduce the oxygenation of the mother's blood to the point where serious damage may be inflicted upon the nervous system of the fetus. A critical period exists during childbirth before the oxygen is supplied by the child's own lungs. The stoppage of the oxygen supply during this period through prolonged labor (asphyxia from pressure) may produce degenerative changes in the cerebral neurones, swellings, hemorrhages, and dead cells. Lack of oxygen also decreases carbon dioxide and increases lactic acid, an excess of which may prove fatal, that is, when the hydrogen ion concentration is reduced below the neutral point. An inadequate supply of oxygen will cause a low brain metabolism. Although the effects of anoxia are most destructive in the newborn, deficient oxygenation in later life tends to produce mental sluggishness and unconsciousness. The restoration of the oxygen supply will restore normal mental functioning unless the frontal lobes have been permanently damaged. In the latter case a permanent mental defect may follow.

According to Lund from 15 to 25 per cent of the newborn suffer from some degree of asphyxia.³⁴

The results of the studies of the relation of difficult deliveries to mental defects, are, it must be admitted, somewhat

³³ Cole, Wyman C., and Kimball, David C., "The Causes and Significance of Asphyxia of the Newborn," *Journal of the Iowa State Medical Society*, 1940, 425-430.

³⁴ Lund, Curtis J., "Prevention of Asphyxia Neonatorum," *American Journal of Obstetrics and Gynecology*, 1941, 931 f.

discrepant. Hanna concluded from a study of 399 cases of difficult births with asphyxia and 206 normal births that no more mental defectives were found among the difficult than among the normal deliveries.³⁵ Dayton did not find any correlation between prolonged labor (over 24 hours for mothers of firstborn and 12 hours for multiparae), instrumental deliveries (low forceps excluded), and unusually difficult labor (as evidenced by tears and lacerations reported by the mothers five to sixteen years later) and ten-point I. Q. groups, except for the groups 0 to 29 and especially 90 plus. "It appears that children of dull normal or low normal intelligence are more likely to be influenced by abnormal birth conditions than children who are definitely in the imbecile, moron, or borderline groups."³⁶ It must be borne in mind that the diagnoses of the birth conditions were based upon the data recorded in the history blanks, which are none too accurate. The association between abnormal labor and skeletal and neurological defects was more definite.

In a follow-up investigation of 19 children with severe asphyxia at birth but without physical signs of injury at the time of delivery according to the hospital records, it was found that all the severely asphyxiated were significantly retarded in mental development when examined from two to eleven years later. The average I. Q. of the asphyxiated was 88 compared with 101 for the controls (siblings or parents). Four had I. Q.'s of 70 or less.³⁷

In an investigation of 132 children of varying degrees of anoxia at birth, Preston classified 97 as of average or higher intelligence, 23 as dull or borderline, and 35 as subnormal.³⁸ The earlier the abnormalities were recognized, the greater was the likelihood that subsequent behavior disorders could

³⁵ Quoted from Shrubsall, Frank C., and Williams, Alfred C., *Mental Deficiency Practice*. London: The University of London Press, 1932, p. 34.

³⁶ Dayton, "Abnormal Labor as an Etiological Factor," p. 404.

³⁷ Darke, Roy A., "Late Effects of Severe Asphyxia Neonatorum: A Preliminary Report," *Journal of Pediatrics*, February, 1944, 148-158.

³⁸ Preston, M. I., "Late Behavior Aspects Found in Cases of Prenatal, Natal, and Postnatal Anoxia," *Journal of Pediatrics*, April, 1945, 26:353-366.

be prevented. The personality of the children thus injured was susceptible to improvement through proper understanding and education.

In a study of 99 infants with cerebral atrophy, probably of extrinsic rather than genetic origin, many attributed to anoxia, Faber classified 74 per cent as mental deficient, 43 per cent as cerebral spastics, and 48 per cent as subject to convulsions. Pediatricians have "been thinking etiologically too much in terms of intracranial hemorrhage and too little in terms of anoxia."³⁹

Schreiber studied the birth records of several hundred mentally deficient children under ten in the Children's Hospital in Michigan and in private practice. After excluding all those suffering from infection after birth and from brain injuries, he found that 70 per cent of the others (252 cases) had a history of anoxia at birth. The birth records of 146 Detroit school children of low I. Q. (45 to 85), after 24 had been excluded because of family histories of low intelligence and 22 because of postnatal trauma or infections, were compared with those of 100 well-adjusted pupils. Compared with the normals, the low I. Q. group had more cases of premature, breech, precipitate, and twin deliveries, and four times as many cases of difficult breathing at birth (apnea) and also of difficult breathing with analgesia (which is indicative of drug-produced insensibility).⁴⁰

With our newer knowledge of the effects of anoxia on nervous tissue, it is apparent that some mental deficient, classified as hereditary cases have, in reality, been brain-injured from natal asphyxia. Unrecognized birth traumas from lack of oxygen are probably responsible for the dullness of some

³⁹ Faber, Harold K., "Cerebral Damage in Infants and in Children: Some Observations on Its Causes and the Possibilities of Its Prevention," *American Journal of Diseases of Children*, July, 1947, 1-9.

⁴⁰ Schreiber, Frederic, "Mental Deficiency from Para-Natal Asphyxia," *Journal of Psycho-Asthenics*, 1939, 7:95-106; "Cerebral Anoxia at Birth," *Journal of Exceptional Children*, 1943, 227.

children who without such injuries would have rated as normal.

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POSTNATAL FACTORS. These factors are of importance chiefly during the early years of life.

Infectious diseases are the chief causes of mental deficiency after birth, most important of which are cerebrospinal meningitis, encephalitis lethargica (popularly called sleeping sickness), policephalitis, followed by poliomyelitis (infantile paralysis), and possibly chronic rheumatic infection when it causes chronic encephalitis.⁴¹ Less important are measles, whooping cough (particularly in infancy),⁴² diphtheria, scarlet fever, and other childhood infections.

Glandular dysfunctioning: of undoubted importance in the production of certain special types.

Nutritive disorders or malnutrition: of undetermined influence.

Long continued and severe epileptic seizures, especially of the grand mal type: unchecked seizures can produce mental

⁴¹ Benda, Clemens E., "Ten Years Research in Mental Deficiency," *American Journal of Mental Deficiency*, October, 1946, 181 f.

⁴² For a recent study see Levy, Sol, and Perry, H. A., "Pertussis as a Cause of Mental Deficiency," *American Journal of Mental Deficiency*, January, 1948, 217-226.

deficiency in some potentially normal brains and in a larger number of the nervously unstable types and can reduce the mental level of the mentally deficient.

Head injuries through falls or blows: very rare.

Sense deprivation, blindness or deafness in the normally endowed: of importance only if the victims have been denied orthogenic instruction and adequate cultural stimulation.

Lack of cultural stimulation, social contacts, and educational advantages: probably far more important in the production of personality and social maladjustments, educational retardation and deficiencies, and mental dullness than genuine (not pseudo) mental defectiveness.

Space does not permit even a brief summary of the numerous investigations bearing on the role of the factors enumerated above or on other hypothetical causal agencies. Reference must be confined to a few studies of malnutrition after birth, all of practical significance for the development of healthy morale and psychosomatic efficiency.

One of the most interesting studies of the effects of prolonged malnutrition on the development of normal and mentally retarded children is reported by Smiley Blanton at the close of World War I. The medical corps of the U. S. Army of Occupation investigated 6,500 school children, ages five and a half to fourteen, in Trier, Germany. 40 to 50 per cent of whom had suffered from obvious malnutrition for from two to three years. Blanton's general conclusion from this investigation was:

Children free from organic nervous disease, and with parents of average intelligence, very rarely become feeble-minded through malnutrition, even of an extreme degree. One of the most important things shown in this whole study is how the nervous system of children of good nervous stock can resist malnutrition of an extreme degree extending over three years. But the feeble-minded, the borderline defectives, and those classed as dull . . . are affected, and often permanently so, by malnutrition of even a moderate severity. . . . The feeble-minded drop to lower levels

of intelligence. The borderline defectives become like the definitely feeble-minded, and the normally dull children become like borderline defectives.⁴³

Kugelmass studied the effects of nutritional improvement on the mentality of 182 children from two to nine years of age, equally divided between institutional and out-patient cases. In group I, 41 retarded and 50 normal children were malnourished at the first test, but well nourished at the second test given from about seven months to several years later. The retarded group showed a mean gain of 10 I. Q. points (between the Kuhlmann-Binet and Stanford-Binet, which are not identical) and the normal group 18 I. Q. points. The younger children improved more than the older ones. In a comparable check group of well-nourished children at the time of the test, the retarded showed no change and the mentally normal a -0.9 change in the second test. Improved nutrition in the case of the malnourished normal children resulted in greater interest, responsiveness, and attentiveness and the alleviation of nervous symptoms.⁴⁴

The intravenous injection of large doses (6.7 milligrams) of vitamin B₁ (thiamine chloride, the beri-beri prevention vitamin) at three- to four-day intervals for three months (about three times the adult maintenance requirement)⁴⁵ did not produce any mental improvement in eight young mental defectives. In another experiment 2 milligrams of thiamine a day given to children during a three-month period did not reveal any significant improvement in height, weight, school attendance, or performance in school examinations.⁴⁶

⁴³ Blanton, Smiley, "Mental and Nervous Changes in the Children of the Volksschulen of Trier, Germany, Caused by Malnutrition," *Mental Hygiene*, July, 1919, 343-386.

⁴⁴ Kugelmass, Isaac N., "The Nutritional Basis of Nervous and Mental Disorders in Children," *American Journal of Mental Deficiency*, October, 1943, 142-152; *American Journal of Digestive Diseases*, November, 1944, 11:268-273.

⁴⁵ Lewald, James, and Alexander, Eugene J., "A Report on Thiamine Chloride (Vitamin B₁) in Mental Deficiency," *Journal of Psycho-Asthenics*, 1939, 2:34-39.

⁴⁶ Mangold, S., "Thiamine for School Children," *Medical Journal for Australia*, 1945, 1:34.

On the other hand, a controlled experiment in which 2 milligrams of thiamine a day were administered for a period of a year to a group of 55 children matched with respect to age, sex, height, weight, intelligence test score, educational achievement, and length of residence in an orphanage with another group of 55 children who received placebo tablets showed marked improvement in the thiamine squad in visual acuity, reading, memorizing new materials, educational achievement, and in a code substitution test. At the end of the year the administration of the thiamine and placebo tablets was reversed with 20 pairs of the same children. The tests at the end of the year revealed that in most instances the children who had received the thiamine the second year (and the placebo the first year) surpassed their matched companions (who received the placebo the second year and the thiamine the first year). The experiment was conducted during the two years without any knowledge by the tester or by the children regarding the group that received the thiamine or the placebo.⁴⁷

Experiments have also been conducted with the use of vitamin B₂ (riboflavin), benzedrine, and glutamic acid. The administration of vitamin B₂ produced slight physical improvement in a group of nine physically retarded mental defectives with organic brain defects, but it did not result in any mental improvement, as shown by a battery of tests, as compared with the control group of the same life age and height age. To the diet of the experimental group was added one tablet of riboflavin (1 mg., 400 units) "plus a diet rich in vitamin B₂, 1,000 to 1,500 Sherman units" for one month. Thereafter the experimental group received a tablet of riboflavin daily (5 mg., 2,000 Sherman units).⁴⁸

⁴⁷ Harrell, Ruth P., "Mental Response to Added Thiamine," *Journal of Nutrition*, March, 1946, 31:283-298.

⁴⁸ Stevenson, Iris, and Strauss, Alfred A., "The Effects of an Enriched Vitamin B₂ (Riboflavin) Diet on a Group of Mentally Defective Children with Retardation in Physical Growth," *American Journal of Mental Deficiency*, October, 1943, 153-156.

In an experiment on 23 mental defectives who were given daily doses of 30 milligrams of the vaunted intellectual stimulant, benzedrine, for six months, "striking improvement occurred only in 13 per cent" (three primary cases without neurological or endocrine stigmata) with whom, however, the "drug stimulus was associated with intense educational procedures. In these cases the numerical figure for the intelligence quotient does not change. . . . Yet the tests for performance ability showed an average increase of one and one-half years after three months of benzedrine administration." In 22 per cent increased alertness and responsiveness were noted, but no appreciable increase in performance or learning ability was observed. In 65 per cent "no improvement was noted that could not have been achieved by intensive educational measures without benzedrine."⁴⁹ Obviously there appears to be little evidence to justify the optimistic expectation that mental defectives can be restored through benzedrine therapy.

The daily administration of 6 to 24 grams of glutamic acid (an amino acid produced by a chemical breakdown of ingested protein which is resynthesized into new proteins) for six months showed an average increase in Binet I. Q. of 8 points in eight children between the ages of sixteen months and seventeen and a half years. One increased as much as four years. Subnormal children showed the greatest improvement. Personality changes were also revealed in the retest responses.⁵⁰ Glutamic acid is supposed to reactivate the enzyme systems purported to be concerned with the trans-

⁴⁹ Moskowitz, Harry, "Benzedrine Therapy for the Mentally Handicapped," *American Journal of Mental Deficiency*, 1941, 540-543. See also Kleemeier, Lyla B., and Robert W., "Effects of Benzedrine Sulfate (Amphetamine) on Psychomotor Performance," *American Journal of Psychology*, January, 1947, 89-100; Bakwin, Harry, "Benzedrine in Behavior Disorders of Children," *Journal of Pediatrics*, 1948, 32:215-216.

⁵⁰ Zimmerman, Frederick T., Burgemeister, Bessie B., and Putnam, Tracey J., "Effect of Glutamic Acid on Mental Functioning in Children and Adolescents," *Archives of Neurology and Psychiatry*, November, 1946, 56:489-506; "A Group Study of the Effect of Glutamic Acid upon Mental Functioning in Children and Adolescents," *Psychosomatic Medicine*, May-June, 1947, 9:175-183.

mission of neural impulses.⁵¹ White rats, when treated with glutamic acid, learn a maze problem more rapidly.⁵² Definite conclusions regarding the character and permanence of the orthophrenic influences of chemical and vitamin stimulation in children of various ability levels cannot be formulated with certainty without more extensive research.

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Waelsch, Heinrich, "A Biochemical Consideration of Mental Deficiency, The Role of Glutamic Acid," *American Journal of Mental Deficiency*, April, 1948, 305-313.

⁵¹ Nachmansohn, D., John, H. H., and Waelsch, H., "Effect of Glutamic Acid on the Formation of Acetyl-Choline," *Journal of Biological Chemistry*, 1943, 150:485-486.

⁵² Albert, K. E., and Warden, C. J., "The Level of Performance in the White Rat," *Science*, November 24, 1944, 476; Zimmerman, Frederick T., and Ross, S., "Effect of Glutamic Acid and Other Amino Acids on Maze Learning in the White Rat," *Archives of Neurology and Psychiatry*, May, 1944, 51:446-451.

The following article contains many references on the mental ("psychocellular," in terms of the author) effects of vitamins, hormones, chemical elements, and foods—

Clark, Leland C., "The Chemistry of Human Behavior," *American Journal of Orthopsychiatry*, January, 1948, 140-152.

Relative prevalence of hereditary and acquired mental limitations

It is obvious that it is impossible to determine with mathematical exactness the relative proportion of the primary and secondary forms of mental deficiency. The estimates, sometimes little more than shrewd conjectures, will, of course, vary greatly according to the type of mental defectiveness. The sharp division into hereditary and environmental is undoubtedly applicable to certain types and certain cases; other overlapping cases represent the joint influence of both factors in degrees not easily determinable. In some cases no unambiguous causation is discernible. Far more ancestral studies have been made of institutional than of extra-institutional cases of mental defect, and far more of mental defectives than of merely dull or backward children. The following is a brief and concededly incomplete summary of estimates of the proportion of the inherited and acquired types.⁵³

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For a summary of the studies before 1930—

Paterson, Donald G., *Physique and Intellect*. New York: D. Appleton-Century Company, 1930.

Wallin, "Causative Factors of Mental Inferiority and the Prevention of Degeneracy."

Three of the earliest British estimates are by Lapage, Tredgold, and Mercier, at least the first two of whom are recognized medical authorities on the mentally defective. Lapage

⁵³ For a summary of studies of the different physiological factors that may affect intelligence see the Thirty-Ninth Yearbook of the National Society for the Study of Education, *Intelligence: Its Nature and Nurture*, Part I, "Comparative and Critical Exposition," 1940, pp. 93-141.

classified about 90 per cent of the feeble-minded as primary and held that "the acquired conditions, if they have any effect at all, act merely as secondary factors." Tredgold's estimate, submitted in 1905 to the British Royal Commission on the Feeble-Minded, was also 90 per cent. In 1937, basing his work on his "investigation into some hundreds of cases of mental defect of all ages, degrees, and types . . . made some years ago," he scaled the estimate to 80 per cent and admitted that adverse environmental factors of various kinds are present in most cases, sometimes serving as a "contributory, or precipitating factor." The number without discoverable cause are "not more than one or two per cent."⁵⁴ On the other hand, Charles Mercier maintained that "the frequent transmission of feeble-mindedness by inheritance" has not been proved. The following are representative views of later investigators arranged in chronological order.

Goddard—America, 1914, based on the genealogical investigation of 11,389 ancestors throughout several generations of 310 inmates in the Training School at Vineland, N. J.: 54 per cent "undoubtedly hereditary"; 11.3 per cent "very probably hereditary"; 12 per cent had a "neuropathic ancestry"; or a total of 77.3 per cent primary cases; 19 per cent accidental; 2.6 per cent undetermined. A "substratum of hereditary defect" is apparently assumed for almost all cases. Feeble-mindedness in the ancestors is the all-sufficient cause of feeble-mindedness in the child.⁵⁵

Wallin—America, 1922, based on the analysis of the family history data (personally checked from the reports submitted largely by the school nurses from home investigation trips) on 872 consecutive clinic cases examined in 1914 and 1915 in the psychoeducational clinic of the St. Louis department of public instruction, of varying intelligence levels from idiocy

⁵⁴ Tredgold, *Mental Deficiency*, pp. 27 f., 62.

⁵⁵ Goddard, *Feeble-Mindedness, Its Causes and Consequences*, pp. 437 f. 466-468.

to normality: obscure causation (no etiological diagnosis attempted), 74 per cent; for the 229 examinees in whom the causation seemed the clearest, 46.3 per cent hereditary, 37.1 per cent acquired, and 16.6 per cent partly hereditary and partly acquired.⁵⁶

Larsen—Denmark, 1931, based on 1,000 institutional cases, exclusive of a few diagnosed as psychotic, encephalitic, and neurological, but inclusive of mongols, microcephalics, and endocrine dystrophies: 76.2 per cent hereditary.⁵⁷

Shrubsall and Williams—England, 1932, based on 10,000 defective school children: 71.2 per cent inherited, exclusive of mongols, cretins, and microcephalics.

Penrose—England, 1934, based on 513 institutional cases: 29 per cent primary; 9 per cent secondary; 62 per cent unclassifiable as either directly endogenous or exogenous "because the effects of environment and heredity were so very difficult to extricate from one another." "In both low and high grades a slight preponderance of secondary causes may be met with. Among medium grades probably heredity plays a large but not exclusive part."⁵⁸ Forty per cent of the cases studied were classified as morons (according to Yannet).

Doll—America, 1934, based on the inmates in the Vineland Training School: 30 per cent plausibly hereditary, 30 per cent secondary, and 40 per cent unclassified or unknown.⁵⁹ Doll's exogenous category includes "pathological alterations of normal development as well as some relatively rare heredi-

⁵⁶ Wallin, "The Causative Factors of Mental Inferiority, and the Prevention of Degeneracy" (this article contains a summary of other estimates); *The Education of Handicapped Children*, pp. 274 f.

⁵⁷ Larsen, Erik J., "A Neurologic-Etiologic Study of 1000 Mental Defectives," *Acta Psychiatrica et Neurologica*, 1931, 37-51.

⁵⁸ Penrose, *Mental Defect*, pp. 88 f. In his later investigation of 1,280 cases Penrose makes no estimate of the percentage he regarded as primary or secondary.

⁵⁹ *Annual Report* from the Department of Research, Vineland Training School, 1934, 31:112-123.

tary types of pathological morphology represented by the clinical varieties of mental deficiency."⁶⁰ The latter, apparently, is a mixed category.

Rosanoff, Handy, and Plesset—America, 1937, on the basis of studies of mental disorders of various kinds in one or both members of 1,104 pairs of twins of different types of the same and opposite sex: "scarcely more than one-half of the cases of mental deficiency are of hereditary origin."⁶¹ Heredity played a more important role among the high grade than among the low grade cases and among cases uncomplicated with epilepsy, infantile palsy, and allied disorders.

Benda—America, 1943, based on 100 autopsies of idiots and imbeciles (exclusive of morons and mongols) at the Wrentham State School (Mass.), gave the following distribution:

	<i>Antenatal Causes</i>	<i>Natal (mostly asphyxiation)</i>	<i>Postnatal</i>
Idiots	50.3%	33. %	18.4%
Imbeciles	40.0%	9.1%	50.9%

Fifty per cent of the idiots and 40 per cent of the imbeciles are attributed to "developmental failure in the prenatal period, long before the child is born." The 31.3 per cent of idiots and 9.1 per cent of imbeciles ascribed to birth injuries (mostly anoxia) had normal parents and siblings with negative family histories, all were fairly normal physically except for the "neurological handicaps," and the brains in the normal areas showed normal maturation. "Relatively few cases of idiocy develop in the postnatal period. In contrast, however, almost one-half of all imbeciles is caused at that time." "The high grade defectives in state institutions are mainly hereditary cases, and the number of cases where a birth injury

⁶⁰ Doll, "The Essentials of an Inclusive Concept of Mental Deficiency," *American Journal of Mental Deficiency*, 1941, 46:214-219.

⁶¹ Rosanoff, Aaron, Handy, Leva M., and Plesset, Isabel R., "The Etiology of Mental Deficiency with Special Reference to Its Occurrence in Twins," *Psychological Monographs*, No. 216, 1937, p. 130.

or residues of encephalitis are suspected is negligible. On the other hand, private institutions have quite a number of accidental cases of high grade mental defectives." The microscopic study of 12 so-called "functional cases" of mental deficiency "revealed striking pathology."⁶² In his later study of 200 autopsies, Benda classifies 35 mental defectives of various levels as endogenously produced "morbid entities."

Halperin—America, 1945, on the basis of 338 mental deficient in state institutions from northern Ohio (exclusive of epileptics), who were tested by Form L, Terman-Merrill Binet, 150 parents tested by the non-language test used in the British army, and the educational achievement scores, scholastic school reports, and Binet scores obtained for the siblings: classified 26 per cent as neurological (mostly primary) and 45 per cent as acinical. The latter represent "the lower end of the normal curve in the distribution of intelligence test scores." "There is a noticeable tendency for idiots and imbeciles to come from average homes, as compared to morons who tend to come from inferior and very inferior homes."⁶³ No pedigree histories were compiled in this study, and the tabulation was incomplete from the point of view of genesis.

Yannet—America, 1945, grouped 44.6 per cent of 1,330 consecutive admissions to the Southbury Training School in Connecticut as hereditary cases, 7 per cent as acquired, and 48.4 per cent as of unknown causation. Heredity was considered to be the sufficient cause of 85 per cent of morosity, 31 per cent of imbecility, and only 5.5 per cent of idiocy. The causation was recorded as unknown for only 12 per cent of the morons, as against 61 per cent for the imbeciles, and 84 per cent of the idiots. The mental ability classification

⁶² Benda, C. E., "Prevention of Mental Deficiency from the Viewpoint of Neuropathology, with Special Reference to the Frequency and Significance of Birth Injuries," *American Journal of Mental Deficiency*, July, 1943, 33-45. See also Benda's more recent summary, "Mental Deficiency," in *Progress in Neurology and Psychiatry*, Spiegel, E. A. (editor). New York: Grune & Stratton, 1948, III, pp. 483-501.

⁶³ Halperin, Sidney L., "A Clinico-Genetical Study of Mental Defect," *American Journal of Mental Deficiency*, July, 1945, 8-25.

was based upon the following Stanford-Binet criteria: morons, 50-75; imbeciles, 20-50; and idiots, less than 20.⁶⁴

The consolidated genetic classification of 7,907 first admissions to public institutions for mental defectives in 1943 gives the following results:⁶⁵

	<i>All Levels</i>	<i>Morons</i>	<i>Imbeciles</i>	<i>Idiots</i>
Familial (hereditary)	28.7	44.0	21.2	12.4
Undifferentiated	23.0	29.1	22.8	19.0
Unknown	22.7	11.8	20.4	15.1

The tendency with the passing years has been to attribute fewer cases to heredity, to find more hereditary cases among the higher grades (more among the morons than among the imbeciles and more among the imbeciles than among the idiots), and to classify a considerable number as "undifferentiated" or of "unknown" origin.

Nature of the neuropathic taint antecedent to mental deficiency

Very divergent views have been held regarding the nature of the ancestral defects responsible for mental deficiency in offspring. Hamilton C. Marr held that "the inheritance is chiefly insanity," and Goddard held that feeble-mindedness is the only hereditary antecedent of feeble-mindedness. Penrose considers that the important thing to look for is "inheritance on Mendelian lines," and that "it is far more useful to have accurate knowledge of the sibship and parents of the patient alone than to have any amount of information about cousins and grandparents."⁶⁶ On the other hand, Lapage held that epilepsy, feeble-mindedness, and insanity are largely interchangeable, although the descendants are more likely to have the same defect. Tredgold also maintains that prac-

⁶⁴ Yannet, Herman, "Diagnostic Classification of Patients with Mental Deficiency," *American Journal of Diseases of Children*, August, 1915, 83-88.

⁶⁵ *Patients in Mental Institutions*, 1943.

⁶⁶ Penrose, *Mental Defect*, p. 60.

tically any neuropathic manifestation may be an hereditary antecedent of mental defectiveness although it is commoner for the ancestors of defectives to suffer from such conditions as insanity, epilepsy, dementia, or allied psychopathological states than it is for them to be actually mentally deficient. In the author's genetic investigation the liberal Tredgoldian view was followed with respect to considering any grave neuropathic disorder as a predisposing hereditary factor.

One of the most striking results revealed in the figures cited is the similarity in the findings of Penrose and Wallin who, although employing different techniques in their investigations, left most of their cases undetermined from the standpoint of the nature of the etiological diagnosis, because of the paucity, complexity, or ambiguity of the causal data. Lewis's subcultural group, including 45 per cent of his cases, might also be construed as belonging to this indeterminate category. As already noted, 48.4 per cent of Yannet's cases were not classified etiologically. Some students of mental defect, especially those of an earlier generation, have left no cases undetermined or only a negligible number. The wide differences of opinion between investigators point to the need for further research on the problem of causation.

Many British and American investigations have led to the conclusion that only a minor number of parents and siblings of defectives are mentally defective. According to Shrubsall and Williams only 5 per cent of the fathers or mothers of children in the London special schools were mentally defective, 50 per cent were subnormal or unstable, 35 per cent were of average intelligence, and nearly 10 per cent were slightly above average. Fifteen per cent of one or both parents of Manchester special class pupils were mentally defective, according to Herd. Penrose, who investigated the causation of 1,280 patients in the Royal Eastern Counties Institution in Colchester, England and who frankly acknowledges the uncertainties of genetic diagnoses, concluded that only from 7 to 9 per cent of the parents and siblings of the patients were

mentally defective, with more mothers than fathers defective, and that 13.7 per cent were dull or borderline.⁶⁷ The American figures tend to run somewhat higher. Myerson classified 29.3 per cent of one or both parents of his defectives as feeble-minded and 37.1 per cent as feeble-minded, epileptic, or insane. Halperin, on the basis of psychological tests, classified 16 per cent of the parents of his cases as defective, 41 per cent as inferior, 38 per cent as average, and 5 per cent as above average, and 16 per cent of the siblings were diagnosed as defective. Twelve per cent of one or both parents of children examined in a traveling clinic in Massachusetts were feeble-minded and 6 per cent were insane or epileptic, according to Dayton. The corresponding figures for parents of inmates in the Wrentham State School were 27 per cent and 12 per cent.⁶⁸

During a twelve and a half year period only 7.9 per cent of 1,969 transfers to the St. Louis special schools were siblings. Of the parents who brought the children to the psychoeducational clinic a comment made in 1920 bears repeating: We "venture the statement that of the thousands of parents who have brought children to us for examination, certainly not more than 20 or 30 could have been committed to an institution as feeble-minded. However, many parents have not come to the clinic. Had we seen all of them, the ratio might have been larger."⁶⁹

Although only the minority of parents of mental defectives are themselves mentally defective, numerous surveys by means of standardized tests of intelligence and educational achievement have shown that the average mentality of the offspring correlates positively with the average socio-economic status of the parents, which also correlates more or less closely

⁶⁷ Penrose, Lionel S., *A Clinical and Genetic Study of 1,280 Cases of Mental Defect*. London: His Majesty's Stationary Office, 1938, pp. 20, 30.

⁶⁸ The data from Herd, Dayton, and Myerson are cited from Tredgold, *Mental Deficiency*, p. 28, which also gives additional figures from Germany.

⁶⁹ Wallin, "The Problem Confronting a Psycho-Educational Clinic in a Large Municipality," *Mental Hygiene*, 1920, 103 f.

with their intellectual capacity. It would carry us too far afield to attempt to summarize the relevant data on this question.

Additional consideration will be devoted to the question of heredity in connection with the discussion of the social and eugenical implications of mental defectiveness in another book.

Shortcomings of the pathogenic classification

(1) We cannot in our present state of knowledge always be certain of the mode of origin of many cases of mental impairment, even after a thorough genetic investigation. This circumstance, instead of being a cause for despair or resignation, should serve as a challenge for further fundamental etiological research. We cannot expect to institute effective programs of prevention until the causative factors are fully understood. Parents are prone to seize upon some environmental factor as the adequate cause of a child's deficiency—a fall, a fright, or a slight infection—often primarily as a mechanism of defense against hereditary implications; but an environmental factor may have been only a precipitating circumstance, or a complicating or contributing incident to an underlying constitutional nervous instability or weakness.

(2) The pathogenic classification does not give any precise clue to the mental level of the child. High, medium, and low grade cases exist among both the primary and the secondary types. The gratuitous assumption sometimes made that the "inborn" cases are low grade and should therefore be excluded from the public schools, whereas the acquired cases are high grade, is often the exact opposite of the truth. The victims of serious natal or postnatal brain lesions (traumatic or infectious) often test very low and achieve little intellectual progress or educational advancement. In spite of this shortcoming, the discovery of the cause sometimes indicates the nature of the needed treatment, as in the case of backwardness or emotional instability due to syphilis or serious

malnutrition, or mental defect produced by lack of thyroid secretion in early life. Treatment that is not based on the discovery of the underlying causation usually remains on the symptomatic level and, at best, exerts merely an adjuvant influence.

NATURE OF THE STRUCTURAL DEFECTS IN THE BRAIN TISSUE

This simplified and condensed exposition of neuropathology aims to give an over-all picture of the essential anatomical basis of mental defectiveness without too much involved technical neurological detail of interest primarily to the neurological expert. Although the great variety of bodily divergencies or defects found in the various grades and types of mentally deficient children cannot be expressed in a simple formula, the important fact to remember is that the seat of the essential pathology of mental deficiency resides in the central nervous system. This is true even of the comparatively infrequent cases in which the origin of the defect is in some other organ of the body—such as the thyroid gland in cretinism and the pituitary gland in pituitary infantilism. Any part of the central nervous system may be involved, such as the cerebrum, the cerebellum, the pons, the medulla oblongata (or bulb), the basal ganglia, and the spinal cord.

By way of a very brief review, the central nervous system, the brain and the spinal cord, consists, in barest outline, of the following main parts.

(1) *The cerebrum* or forebrain, the larger part of the brain, is divided into two hemispheres located under the skull and above the brain stem. It consists of an outer layer (called cortex) and five masses of nerve fiber beneath the cortex known as basal ganglia (the extrapyramidal system), which connect the cortex through the brain stem with the nerves in the spinal cord and with the cranial nerves. The brain stem is the continuation of the cord at the base of the brain above the medulla. The motor (or efferent) nerves, which consist of giant pyramid cells and originate in the motor areas of the

cortex, pass through a thick bundle (the "pyramidal tract") and the brain stem and connect with the nerves in the front part of the cord that go to the muscles and glands. The sensory (or afferent) nerves lead from the sense organs through the dorsal region of the cord and the thalamus (one of the basal ganglia, also referred to as the interbrain) to the sensory areas of the cortex. Other regions of the brain contain the association areas.

(2) *The medulla* (bulb) is a thickened continuation of the cord below the pons and in front of the cerebellum. The motor fibers cross in this area, which is an assembly point for fibers that connect different parts of the brain with the cord. Its functions include the coordination of tongue movements in eating and speaking and throat muscles in speaking, and regulation of breathing, digestion, and heart action. Separate centers for breathing and blood circulation have been located on the right and left sides (A. B. Baker, 1946).

(3) *The cerebellum*, a spherical mass of nervous tissue located back of the medulla and somewhat above it, is a center for the coordination of movements and for the maintenance of body balance (in cooperation with the semicircular canals) and muscular tonus. It transmits impressions from and to almost all parts of the nervous system.

(4) *The pons*, a broad bridge of nerve fibers situated in front of the cerebellum, connects one side of the cerebellum with the other. It apparently serves a considerable variety of functions, including mastication, control of facial and eye muscles, throat coordination in speech, and integration of equilibration impulses from the semicircular canals.

Although both the surface and the deeplying areas may be involved in the brain defects of the mentally defective, the essential locus, probably, is the outer layer or mantle of the cerebrum, the cerebral cortex, and, especially, the outer layer of the cortex known as the neopallium (Elliot Smith). Histological investigations (histology deals with the microscopic structure of the tissues) seem to show that this is the

latest portion of the brain to evolve in the history of the race (phylogenesis) and to develop in the history of the individual (ontogenesis). The genetic order of development of the cortex is from the inner to the outer layers. The assumption is that the higher psychic processes—intelligence, judgment, selection, coordination, and adjustment—are connected with the cortex (especially the neopallium), which consists of gray matter (the cell bodies of the neurones) arranged in five layers, according to J. Shaw Bolton, eminent English neurologist. Three of the layers are considered to be of primary importance. These are:

The infragranular layer, which has reached 80 per cent of its adult thickness at birth in the case of human embryos, is supposed to serve instinctive and organic functions. It is the best developed layer in the lower mammals and suffers little reduction in thickness in mental defectives.

The granular layer, which has attained 70 per cent of its adult thickness at birth, is thought to be connected with the sensory and perceptual functions. It is well developed in the higher animals and is reduced in thickness in mental defectives and especially in the victims of sense deprivation—the blind and the deaf.

The supragranular layer (the neopallium), which is only about 50 per cent complete at birth and attains its highest development in man, is thought to be functionally connected with the most complex intellectual processes, especially the nerve cells in the so-called silent areas of the brain, the frontal and parietal lobes (the latter occupy the mid-region on both sides). These lobes contain the so-called "association fibers," which connect the various sensory and motor areas of the cortex. They integrate the impressions derived from the different senses into the meaningful wholes (gestalts) that are at the basis of concept formation and reflective thinking. The supragranular layer is usually diminished in thickness in mental deficient, depending upon the degree of the defect, especially in the prefrontal and parietal areas. This layer is

the least developed in amentia and the first to undergo dissolution in dementia. "It is the last layer of the cortex to be evolved, the last to commence to develop, the last to attain maturity, and consequently the first to undergo retrogression. . . . Amentia is the result of an imperfection of neuronie development which is particularly marked in the granular and supragranular layers of the cortex of the prefrontal and parietal lobes of the brain" (Tredgold). The agenesis or deficiency affects particularly pyramidal neurones which, in the opinion of Bolton, are the substrate for the "associational," "psychic," volitional, and intellectual functions.

Though these findings have usually been accepted, some brain histologists have failed to find any correlation between the number of nerve cells in the granular and supragranular layers and the degree of mental deficiency. Because of the obscurity that shrouds our understanding of the normal and abnormal functioning of the mechanism of the brain and the wide gaps that still exist in our knowledge of the neuropathology of mental deficiency, many of our conclusions regarding the exact relations of definite brain defects and mental defects must be tentative. Many discrepancies still exist in the reported findings of brain histologists, for a variety of causes—differences in the techniques employed, difficulties of interpretation of the multiplicity of the defects sometimes found (that is, their relation to the particular mental abnormalities found), and the large differences that actually exist between cases that may exhibit symptomatic similarities. Moreover, however important the discovery of local cerebral lesions may be for an understanding of mental defects, it must be remembered that the "cartography" or "localization" of brain functions cannot be too sharply drawn, that the different brain areas do not function independently of one another, that the whole brain acts at all times as a unit, and that defects in one area interact upon other areas.

The histological investigations by the pioneers of the brain anatomy of mental defectives, such as Desire Magloire

Bourneville in France, Karl Hammarberg in Sweden, Tredgold, Lewis, and Bolton in England, Alfred W. Wilmarth in the United States, and later investigators, particularly in America, have at least laid the groundwork for a science on the brain anatomy of the mentally defective. Although certain defects exist in the brains of mental defectives which may be regarded as typical, it must be borne constantly in mind that not all defects are found in any one case and that defects that may be found in a given case may not have any causal relation to the symptoms.

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Although the difference in the brain defects between the lowest and the highest grades of mentally deficient children in the preponderance of cases is largely one of degree, a baffling variety of anomalous conditions is revealed by post mortem examinations of the brains of both the primary and the secondary forms. The cerebral defects can be grouped, however, into two main categories: macroscopic or "gross"

lesions, largely perceptible to the naked eye of the brain surgeon, and sometimes to the eye of the lay observer; and microscopic imperfections in the neurones which are so minute that they can only be detected, if at all, by powerful microscopes or appropriate chemical stains.

Macroscopic defects. These gross defects, largely restricted to the brains of the low grade defectives (idiots and imbeciles), are caused either by incomplete development or non-development of the whole or parts of the brain (encephalon)—technically denominated agenesis or hypoplasia—or by processes of degeneration or atrophy which result from infectious diseases, meningeal or cerebral hemorrhages, brain growths, mechanical injuries, and birth asphyxia.

These cerebral defects assume a great variety of forms. (a) Stunted development of the brain in whole or in part. for example, one hemisphere (hemiatrophy) or the frontal lobes. There is, on the average, a slight correlation between the size of the brain (cranial capacity) and intelligence;⁷⁰ the averages for idiots and imbeciles are perceptibly smaller than for normal persons of the same age. But there are many exceptions to the rule: some persons with very small heads are bright and clever, whereas others with abnormally large heads may be notably stupid or feeble-minded. Though the size of the brain is not a negligible factor, it is of minor importance compared with the quality or sensitivity of the neurones. Accordingly, it should not be surprising to learn that a gross brain lesion is not incompatible with fairly normal mentality.

(b) Paucity or simplicity of the development of the folds of the cortex, called convolutions or gyri (known as microgyria). This defect is particularly notable in the brains of idiots, in the first instance, and also of imbeciles. The more richly convoluted the brain is, the more extensive is the corti-

⁷⁰ For the results of investigations along this line see Penrose, *Mental Defect*, pp. 24-33 and the references there; Doll, *Anthropometry as an Aid to Mental Diagnosis*. Vineland, N. J.: Publications of the Training School, No. 8, 1916.

cal area, which is vitally related to the higher forms of intelligence.

(c) The existence of clefts, cavities, or pores (porencephaly), other than the natural ones (ventricles), as a result of a congenital deformity, lack of development (agenesis), vascular occlusion, and traumatic or inflammatory lesions. These cavities often develop in the region of the sylvian fissure and may extend into the lateral ventricle.

(d) Enlargement of the ventricles, frequent in the birth injured.

(e) Accumulation of cerebrospinal fluid, which may produce hydrocephalus.

(f) The growth of cysts (sacs containing fluid) in degenerating or sclerosing tissue, a condition sometimes amenable to surgical interference.

(g) Adhesion of brain membranes (called meninges) to the cortex, as a result of meningitis.

(h) Cerebral tumors. The infantile form, very rare, proves fatal before the mental defect is noticeable; the tuberous form is discussed under (i).

(i) Growth of areas of hardening or induration, known as sclerosis (or gliosis), as a result of developmental anomalies, or inflammatory processes, or vascular lesions before or during birth which cause hemorrhages and degeneration of the nerve cells and an excessive development of non-nervous supporting tissue (called neuroglia or glia cells). In the sclerotic areas the neurones are atrophied and functionless. Wilmarth found sclerosis to be the most prevalent brain defect, affecting 25 per cent of the 100 brains he examined of feeble-minded inmates at the Elwyn Training School, Pennsylvania (the largest number of autopsies on such brains in America until within recent times). The sclerosis occurs in two forms, diffused and circumscribed or localized.

In the diffuse form (often discovered only in post mortems) the sclerosis is widely scattered throughout the brain and may lead to an enlargement ("hypertropic sclerosis") or diminu-

tion ("atrophic sclerosis") of the brain. The contraction usually follows the initial enlargement. The atrophic form, very rare, is characterized by muscular tremors that affect the whole body and resemble paralysis agitans or affect only the head (shaking); muscular weakness, with stiffness of the arms and legs other than paralysis; epileptic convulsions in rare instances; and severe amentia with progressive deterioration.

The hypertrophic form, also relatively rare, is characterized by an increase in the size and density of the brain from the proliferation of the neuroglia tissue. The largest recorded head circumference of a hypertrophic macrocephalic (*makros*, large) head is 28 inches (Tredgold) and the greatest weight somewhat over 62 ounces (Daniel Brunet), the medium weight of the normal adult male brain being about 48 ounces and of the female brain about 44 ounces. The term macrocephaly is applied to excessively large heads irrespective of the type; that is, whether they belong to the sclerotic, hydrocephalic, or any other kind. The massive head associated with sclerosis is squarish rather than round, with a prominent, vertical forehead and conspicuous frontal prominences, and with the greatest circumference at the level of the superciliary ridges (at the eyebrows) and not at the level of the temples, as in the case of hydrocephaly. There is no bulging of the fontanelle or sutures or such long continuing enlargement of the head as is frequently found in hydrocephaly. Severe headaches, general muscular weakness with unsteady gait and feeble grasp, and epileptic convulsions are the prominent traits found in this form of brain defect. Although often intelligent looking, with prominent foreheads, the patient has a brain enlargement which represents so much excess baggage—an overgrowth of dense, inert neuroglia with atrophied neurones—and the victims prove to be mentally deficient in varying degrees, depending upon the extent of the neuroglia and the frequency and severity of the convulsions. The seizures may reduce a moron to an idiot

and bring fatal termination in early life. The mild cases may survive into late adulthood. The milder forms, and the more severe forms if the seizures abate or can be controlled, are amenable to training in accordance with the level of intelligence. Ordinarily sclerotics are good-natured and placid when not suffering from headaches or seizures. Some of them have unusually good memories for dates and numbers. Possibly the incidence of sclerotics can be reduced somewhat by greater care in the prevention of natal anoxia (oxygen deprivation).

In the circumscribed or localized form of sclerosis hard, pale or pearly white nodules varying in size from a pea to a walnut are found chiefly in the cerebral cortex, especially in the frontal lobes, on the walls of the lateral ventricles, and in the basal ganglia. A reduction of nervous tissue and increase in neuroglia occur in the region of the nodules. Changes are noted in the form and shape of the ganglion cells (large nerve cells found especially in the spinal ganglia). Giant cells may be found two or three times as large as the pyramidal cells, which are large, multipolar, pyramid-shaped ganglion cells of the cerebral cortex. Tumors may also develop in the kidneys, heart, thyroid and thymus glands, and other organs. This kind of gliosis (development of neuroglia in tumors) was named *tuberous sclerosis* by Bourneville in 1880 and *epiloia* by Edward B. Sherlock in 1911. This rare condition occurs in families with a high incidence of psychotic conditions (mental derangements) and, although the etiology is obscure, it is believed to be of germinal origin.⁷¹ According to John Thompson, the condition probably dates from the seventh fetal month. It rarely affects more than one person in the family. Although a number of clinical types have been identified, the three commonest features of the symptom complex are a characteristic skin rash, epilep-

⁷¹ A Mendelian dominant, according to Penrose. A dominant is a character that appears in all the hybrid offspring to the exclusion of the contrasting character present in the other parent. See page 206.

tic convulsions, and mental defectiveness. The skin rash, which appears about the fourth or fifth year and which constitutes the best means of diagnosing the condition during life, is known as *adenoma sebaceum* (or Pringle's disease).⁷²



FIG. 4. *Adenoma Sebaceum* (butterfly rash), characterized by reddish-yellow nodules, mainly on the skin near the sides of the nose.

It consists of a collection of reddish-yellow papules or nodules distributed mainly on the forehead and cheeks. The distribution on the sides of the nose roughly resembles the shape of a butterfly, hence the disorder is also called "butterfly rash." (See Fig. 4.) The skin eruption occurs in about 50

⁷² For a recent discussion of the condition consult Duffy, Ruth E., "Tuberous Sclerosis—Report of One Case," *American Journal of Mental Deficiency*, January, 1945, 261-265. See also Globus, Joseph H., "Malformation in the Central Nervous System," in *Cytology and Cellular Pathology of the Nervous System*, Penfield, Wilder (editor) New York: Paul B. Hoeber, 1932, pp. 1150-1166.

per cent of the cases and possibly more because this rare condition is undoubtedly sometimes misdiagnosed as acne (*acne rosacea*). There is no cure for this disfigurement, although attempts have been made to remove the nodules by electrical coagulation and gross dissection. The major and minor forms of epileptic seizures, in varying degrees of severity, occur at varying intervals from the first year until death supervenes, usually before maturity is reached, sometimes in attacks of *status epilepticus*, although some tuberous sclerotics suffer very few attacks or none at all. Cases of epiloia have been found without intellectual impairment, but most of them undergo progressive deterioration (possibly as a result of the seizures) and classify as idiots or imbeciles. The mental defect is ordinarily present from early life, as shown by the slowness with which they learn to sit up, stand, walk, and talk. Some never acquire speech. Some eventually develop a mental disorder resembling dementia praecox. Some can be trained to perform simple tasks, but most are not amenable to educational therapy. No data are available on the number of high grade, unidentified cases that may be enrolled in public schools. Nothing is as yet known about the prevention of the condition. The newer forms of anticonvulsant drugs are apparently ineffective with epileptic sclerotics.

Microscopic neurone defects. The microscopic defects in the cerebral neurones constitute the essential basis of mental deficiency in the majority of mentally defective children. They are the only kind of defects discoverable in the brains of most of the high grade defectives. The chief defects include:

(a) Numerical deficiency of neurones, especially, according to some neurologists, in the supragranular layer, and, to a less extent, in the granular layer, especially in the prefrontal and parietal lobes as far as concerns the high grade cases, and in all parts of the brain for the low grade cases. Hammarberg found a high correlation between the number of pyrami-

dal cells and the degree of mental deficiency. Bolton found that the depth of the pyramidal layer of the prefrontal region varies directly with the degree of mental deficiency.

(b) Imperfect development of the neurones, first noted by Bevan Lewis in 1879. Many neurones never attain full maturity, but remain in various stages of immaturity. Some remain in the embryonic stage (neuroblasts), globular masses without a definite cell body and without the processes (neurites and dendrites) which convey the nervous impulses through the synapses from one neurone to another. Such immature neurones, which are functionless, are also found in normal brains but in a much smaller ratio.

(c) Some nerve cells contain much pigment, especially in the deeper pyramidal layer, which may indicate defects of metabolism and degenerative changes. Chapter 13 will consider various kinds of mental defectiveness associated with biochemical and metabolic disorders of the neurones.

(d) The arrangement of the cells (especially the pyramidal ones) may be haphazard. The cells may lie obliquely, upside down, or horizontally instead of at right angles to the surface. Hammarberg, however, maintains that the irregularity is no greater than that found in the brains of normal persons.

(e) Proliferation of new processes which, however, do not serve any neural function and represent so much "excess baggage."

Limitations of the pathological classification

It is very difficult to discover the exact brain pathology during the life of the individual. The post mortems may yield valuable results for science, but are of no benefit to the deceased. Trephining the skull is a very inconvenient operation to the patient and is contraindicated in the large majority of cases, as no benefits accrue from the operation. The X-ray technique called encephalography (or, better, pneumo-encephalography), devised by Walter Dandy in 1918, will

reveal cavities and areas of gross destruction.⁷³ In this method the spinal fluid, which surrounds the brain and fills the ventricles, is drained off by a lumbar puncture, and air is injected with a syringe into the ventricles and spaces between the convolutions. All the spaces filled with air appear



FIG. 5. Pneumoencephalogram of a normal brain, left lateral view. The air is shown as dark streaks between the convolutions and as a dark half moon in the lateral ventricle.*

as dark shadows on the plate (encephalogram). In Fig. 5 the small dark lines show the air between the convolutions; the dark half-moon area is the air in the lateral ventricle. Fig. 6 is the pneumoencephalogram of an eleven-year-old girl who developed epilepsy eight months after an automobile

⁷³ Davis, David B., "Encephalography—The Method and Its Use in Mental Deficiency," *Journal of Psycho-Asthenics*, 1939, 2:72-78.

* From Davis, David B., in the *Journal of Exceptional Children*, April, 1939, p. 171.

accident. The encephalograms demonstrated that her grand mal convulsions were caused by a brain injury and that the earlier tentative diagnosis of idiopathic epilepsy was wrong. The darkened areas reveal a tear in the posterior part of the brain and an enlarged lateral ventricle (the left, shown by

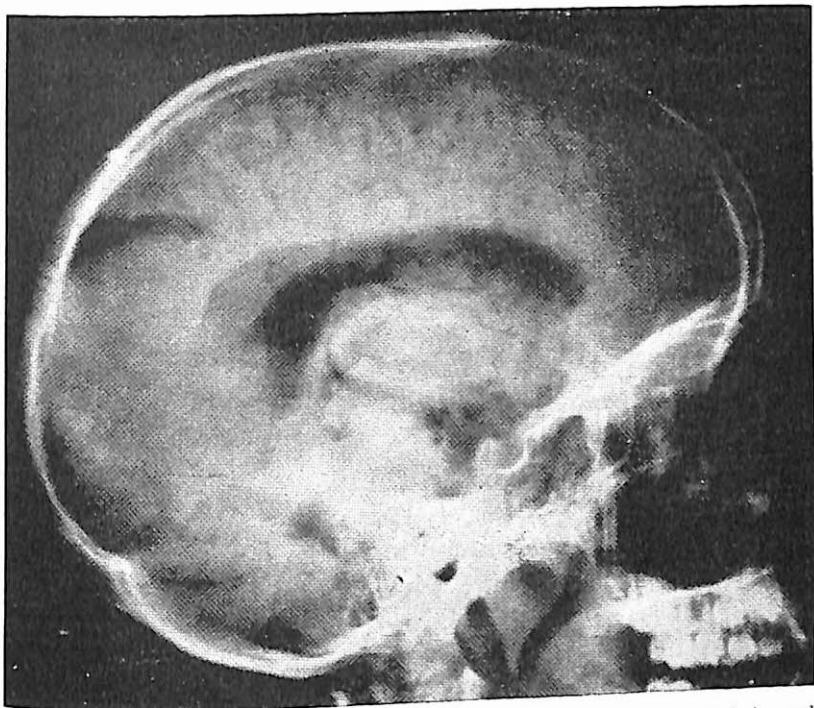


FIG. 6. Pneumoencephalogram of a damaged brain in an epileptic, left lateral view. Large destruction of tissue is shown in the posterior part and in the ventricle (black areas).*

another view). The dilatation of the ventricle was caused by the brain destruction. The encephalograms revealed that in this case surgery could not remove the lesions, nor could the condition be cured by medicine, although one of the modern anticonvulsant drugs might lessen the attack.⁷⁴

* From Davis, *ibid.*, p. 172.

⁷⁴ The plates are reproduced with the permission of Davis, "The Epilepsies," *Journal of Exceptional Children*, April, 1939, 166-175, 178.

Another new method, devised by Hans Berger of Germany in 1929, consists in the amplification about a million times of the electrical waves generated by the brain. The waves are transmitted from electrodes (metal plates) attached by adhesive tape to various parts of the skull through a vacuum tube amplifier to an oscillograph which records the waves on a moving ribbon of paper. (See Fig. 19, and the further discussion in Chapter 14). Such tracings are referred to as electroencephalograms. The machine, which looks something like a radio, measures very slight variations in the rate and intensity of electrical impulses originating in the cortex. The intensity (amplitude) of the waves varies from 10 to 50 millionths of a volt. The frequency in normal adults is about 10 waves a second. These waves are known as alpha waves. The rate is slower and more irregular during sleep than during waking hours. It is increased by exercise and excitement. It varies with age. This technique has added immeasurably to our knowledge of seizures and allied states. How much light it will throw on the brain condition of the different grades and clinical types of the mentally defective is a challenging problem for investigation. From the limited studies made thus far it appears that cretins, for example, have a low frequency rate, and the "voltage of the brain waves found in hydrocephalics is almost twice as great as the average for the mentally normal group." The waves apparently aid in the diagnosis of porencephaly and the location of tumors and sclerotic areas.

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There is no invariable or exact relation between the extent of the brain lesions and the degree of intelligence, the nature of the personality peculiarities, or the physical signs and symptoms. Obviously, therefore, the psychological diagnosis of a mental deviate must be based on criteria other than those of brain pathology, even were it possible to obtain a fairly accurate picture of the brain condition of the living client.

Tredgold's broadest generalization is to the effect that there is on the whole a "direct correlation" between the extent of imperfection of neuronie development and the degree of mental defect.⁷⁵ The corresponding conclusion of Fernald, Southard, and Taft, based upon ten autopsies of brains of defectives, is that "at least the brains of least complexity are correlated with the minds of least range, and . . . the brains of greater complexity are in a general way correlated with minds of greater range."⁷⁶ Although these conclusions are doubtless correct, post mortems on brains of mental defectives have shown that some high grade defectives have suffered from gross brain lesions without behavioral betrayal of such defects during life. On the other hand, it has not been possible to find any neural defects whatever in some cases, even low grade cases. Neurones in some idiots have appeared perfectly normal.⁷⁷ Gray's autopsies of 38 unselected cases (19 idiots, 15 imbeciles, 2 morons, and 2 borderline cases) failed

⁷⁵ Tredgold, *Mental Deficiency*, p. 131.

⁷⁶ Fernald, Walter E., Southard, Elmer E., and Taft, Annie E., "Waverley Researches."

⁷⁷ Tredgold, *Mental Deficiency*, p. 124.

to reveal any brain abnormalities in 10 idiots, 6 imbeciles, and in the morons and borderline cases.⁷⁸

About 25 of Wilmarth's 100 brains and 3 of Southard and Taft's 10 brains of defectives gave no evidence of brain disease and looked normal. Both William Ireland and Johann Mierzejewski report brains of low grade cases ("idiots") with perfectly developed histological elements. Penrose does not think that any large proportion of brains with I. Q.'s above 50 would show any "characteristic pathological changes," histologically speaking. He, therefore, is of the opinion that the real abnormality in high grade cases "lies in the functioning of the individual brain cells and their inability to form adequate connections with other cells."⁷⁹ The writer believes that such functional defects might explain some cases of so-called pseudo-feeble-mindedness, but that genuine feeble-mindedness always connotes some structural neurone defect, although the defect may be too minute to be detected by present methods of neurological examination. It is possible that the new electron microscopes, which magnify over 100,000 diameters, and which have made visible many disease viruses hitherto undetected, may bring to light these putative submicroscopic neurone changes. Under such a microscope a human hair is magnified to the size of a California redwood tree. Perhaps the improved neurological techniques of the future may reveal ultramicroscopic defects in the brains, not only of the higher grade mental defectives, but also of children who are merely genuinely mentally backward although clearly not feeble-minded. This suggests another limitation in present-day neurological science. Almost nothing is known about the neurological correlation of backwardness from histological investigations of brains of children classified after adequate psychological examinations as merely intellectually backward.

⁷⁸ Gray, Edward W., "An Anatomical Study of the Brain in the Feeble-Minded," *Journal of Psycho-Asthenics*, 1933, 38:162-171.

⁷⁹ Penrose, *Mental Defect*, pp. 43 f.

In connection with the question of the correlation of cerebral defects with mental defects, reference may be made to the surgical removal of portions of the brain without functional injury.

Psychosurgery. Experiments in "psychosurgery," as it has been called, since 1936 (Egas Monis, of Portugal) have shown that portions of the brain (especially in the prefrontal lobe) can be detached, as it were, without any permanent impairment of the intellect and sometimes with improvement in the personality structure. The operation ("prefrontal lobotomy") consists in severing bilaterally the subcortical fibers (white) connecting the frontal lobe and the thalamus, a mass of gray matter at the base of the brain which is supposed to be functionally connected with the emotions. The thalamus is a sort of physiological battery for "charging" ideas with emotional drive. No part of the brain is removed and the cortical areas are left intact. The implication is that the severing of the neural connections between the intellectual and emotional areas reduces the excitability of the subject, renders him less fearsome and apprehensive, and makes for personality stabilization and efficiency. (See Chapter 4.)

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Chapter 9

CLASSIFICATION ACCORDING TO SPECIAL CLINICAL TYPES—ENDOCRINOPATHIC DISORDERS

Prevalence of special clinical types

The total number of special clinical types—those discussed in the remaining chapters and a few other rare types omitted through necessity—constitutes a not negligible proportion of the whole group of the mentally deficient and retarded. La-page classified about 9 per cent of 784 high grade mentally deficient children admitted to special classes in Manchester, England, as cretins, mongols, microcephalics, hydrocephalics, paralytics, and epileptics; and about 41 per cent of 120 rejects (too defective for admission) belonged to these groups. About 46 per cent of Still's hospital cases (presumably low grade) belonged to these groups. Of 2,774 school children personally examined and diagnosed by the writer on the basis of the synoptic findings in the psychoeducational clinic in St. Louis between 1914 and 1921, 4.7 per cent were epileptics, hydrocephalics, paralytics, mongols, and cretins (Table V).¹ If cases among the epileptics, hydrocephalics, and paralytics not diagnosed as feeble-minded are excluded, these five special types constitute 9.8 per cent of the 994 mental defectives of all grades. They constitute 19.4 per cent of the 342

¹ This old classification is used because time did not suffice for tabulating all the comparable data from the 3,960 Ohio examinees, all of whom were interviewed and diagnosed by the writer. The writer was able to interview and diagnose only a small proportion of the larger array of children examined in his Baltimore, Wilmington, and Delaware clinics. Comprehensive medical, personal, and family histories and school data were available for almost all of the St. Louis, Ohio, and Baltimore examinees. The medical data were very inadequate for most of the Delaware cases.

imbeciles as compared with 4.7 per cent of the 644 morons (Table VI).

TABLE V

Diagnosis of 2,774 Consecutive Children Individually Examined in a Public School Psychoeducational Clinic

INTELLIGENCE DIAGNOSIS

	<i>Boys</i>		<i>Girls</i>		<i>Both Sexes</i>	
	<i>No.</i>	<i>Per Cent</i> ^a	<i>No.</i>	<i>Per Cent</i> ^b	<i>No.</i>	<i>Per Cent</i> ^c
Normal	78	4.0	13	1.5	91	3.2
Retarded	87	4.4	21	2.5	108	3.8
Backward	556	28.5	145	17.5	701	25.2
Borderline ^d	571	29.3	210	25.3	781	28.1
Diagnosis deferred	57	2.9	35	4.2	92	3.3
Mentality undetermined	5	0.2	2	0.2	7	0.2
Morons ^e	390	20.0	254	30.7	644	23.2
Imbeciles	198	10.1	144	17.4	342	12.3
Idiots	5	0.2	3	0.3	8	0.2
Total feeble-minded	593	30.4	401	48.4	994	35.7

SUPPLEMENTARY DIAGNOSES—SPECIAL TYPES

	<i>No.</i>	<i>Per Cent</i>	<i>No.</i>	<i>Per Cent</i>	<i>No.</i>	<i>Per Cent</i>
Speech defectives	395	20.28	150	18.13	545	19.64
Motor aphasia	17	0.87	2	0.24	19	0.68
Delinquents	374	19.2	70	8.46	444	16.00
Visual aphasia	14	0.72	2	0.24	16	0.57
Dyslexia	90	4.62	8	0.97	98	3.53
Visual aphasia and dyslexia	104	5.34	10	1.2	114	4.10
Epileptics	30	1.54	17	2.06	47	1.69
Psychopathic	25	1.28	8	0.97	33	1.18
Unstable and neurotic	141	7.24	53	6.41	194	6.99
Mongolians	12	0.61	11	1.33	23	0.86 ^f
Cretins	3	0.15	5 ^g	0.60	8	0.28
Hydrocephalics					4	0.4 ^h
Paralytics					29	1.4 ⁱ

^a Based on the total number of boys (1,947).

^b Based on the total number of girls (827).

^c Based on the total number of boys and girls (2,774).

^d Includes 79 boys and 46 girls diagnosed as potentially feeble-minded.

^e Includes 94 boys and 82 girls diagnosed as potential morons.

^f Three Mongoloid boys and two Mongoloid girls.

^g Two cretinoid defectives.

^h Based on 900 consecutive cases only.

ⁱ Based on 1,986 consecutive cases only.

Under the intelligence diagnosis the successive groups or classes are arranged in the order of descending intelligence or diminishing intelligence quotients. The supplementary diagnoses are restricted to special types and do not include physical defects or diseases. In the case of re-examined children the final classification has been used. Cases of visual aphasia represent the most serious non-readers, formerly referred to as word-blind. The slighter grades, referred to as dyslexia cases, usually yield to appropriate remedial instruction.

TABLE VI

The Percentage Which the Imbeciles and Morons Among the Special Types Constituted of All the Imbeciles and Morons

	<i>Percentage of all Imbeciles</i>		<i>Percentage of all Morons</i>	<i>Percentage of all Feeble- Minded</i>
Epileptic imbeciles...	6.4	Epileptic morons....	1.4	3.1
Paralytic imbeciles...	4.3	Paralytic morons....	2.1	2.9
Hydrocephalic imbeciles.....	0.9	Hydrocephalic morons.....	0.6	0.7
Mongol imbeciles....	5.8	Mongol morons....	0.46	2.3
Cretin imbeciles....	2.0	Cretin morons.....	0.15	0.8
Total.....	19.4	Total.....	4.71	9.84

It is important to emphasize the fact that when these classifications were made in Europe and in America very little was known regarding certain clinical types and nothing whatever was known, not even by the experts on mental defectiveness, about other special physical types that are given brief consideration in this volume. The recent increase in the incidence of special clinical types in residential institutions reflects, not only this expanded knowledge, but the use of improved diagnostic techniques. Thus Penrose's clinical groups included 75.9 per cent of his 1,280 cases, and the "residual" group included only 24.1 per cent.² Halperin classi-

² Penrose, *A Clinical and Genetic Study*, 1938 survey, p. 33.

fied 54 per cent of his cases as special clinical types,³ and Yannet included over 60 per cent in this category.⁴

Of course the proportion of special clinical and physical types among the general run of psychoeducational clinic school cases and candidates for special educational treatment and persons at large in society is much smaller. Nevertheless, the special types constitute a sizable ratio of the whole group of mental subnormals in the schools, particularly if the borderline clinical types are included and also children of borderline level of intelligence. Some of the special types are found among relatively few of the high grade defectives; and the ratio of the high grade defectives is far greater in society and in the schools than in the state residential institutions. Thus the British Mental Deficiency Committee reported that 73 per cent of the mentally defective children and adults in the six geographical areas investigated in England and Wales (two rural, three urban, and one mixed) were "feeble-minded" (high grade defectives), 20 per cent imbeciles, and 5 per cent idiots.⁵ This contrasts with 48 per cent of dullards and "simpletons" (high grade defectives), 34 per cent of imbeciles, and 17 per cent of idiots among Penrose's institutional cases,⁶ and 40 per cent of morons, 26 per cent of imbeciles, and 33 per cent of idiots among Yannet's institutional inmates.⁷

In the St. Louis psychoeducational clinic only 35.7 per cent were classified as feeble-minded, of whom 23.2 per cent were morons, 12.3 per cent imbeciles, and 0.2 per cent idiots. Some of the lower grades had already been drained into the state colony, and others were transferred in the course of the years.

Obviously, all those who deal professionally with mentally

³ Halperin, "A Clinico-Genetical Study," p. 15.

⁴ Yannet, "Diagnostic Classification," p. 84.

⁵ *Report of the Mental Deficiency Committee*, Part IV (by Edmund O. Lewis), p. 79.

⁶ Penrose, *op. cit.*, p. 15.

⁷ Yannet, *op. cit.*, p. 84.

handicapped children and adults should know something about the numerous special types—their physical, mental, and social characteristics and possibilities, and their causation and medical, psychological, social, and educational treatment.

Value of typology. The study of the special clinical types is always interesting and often yields knowledge of great value for dealing intelligently with handicapped children. A few values of such an orientation are mentioned below.

It often indicates the nature of the pathology—as in the case of cretinism, syphilitic amentia, encephalitis, hydrocephalus, the Fröhlich syndrome, and others.

It sometimes gives a clue regarding the required hygienic, medical, or surgical treatment—as in the case of cretinism, syphilitic infection, epilepsy, poliomyelitis and other kinds of muscle disabilities, certain kinds of hydrocephaly, and others.

A knowledge of the clinical type sometimes gives information regarding a child's mental status—as in the case of mongolism, untreated cretinism, pronounced microcephaly, and several types associated with faulty fat (lipoid) nerve cell metabolism.

It sometimes gives an insight into the child's educational potentials, the nature of the educational program he requires, and the need for social care or supervision.

Even a superficial survey of the special types demonstrates conclusively that mentally deficient and retarded children do not constitute a simple, unitary group or entity who can be satisfactorily classified by some single or simple system of tests that can be easily administered by anyone who is able to comprehend and execute the directions, and who require the same kind of educational, psychological, social, or medical treatment. On the other hand, a condensed review of almost 30 highly varied groups of children who manifest varying ratios of differing degrees of mental subnormality should convey convincing proof that the problems of the diagnosis, classification, and medical, educational, and psychological treatment are highly complicated, and that the statement confidently

made in published articles in the early part of the century to the effect that "nothing else is needed in the great mass of cases than this test" (the Binet scale) for the diagnosis of mental defectiveness was and remains entirely too optimistic. If the inclusion of all of these types in our discussion serves no other purpose than to give the student a vivid picture of the essential complexity of the problem of mental subnormality, it will have accomplished an eminently worth-while purpose. It is not essential that elaborate treatments should be accorded all the special types in order to develop a keen realization of this fact. The space here devoted to certain types is highly condensed, not only because of the necessary limitations of the size of the book, but because these particular types do not create onerous problems, on account of their rarity, as many of the other varieties do for the educator, psychologist, physician, and social worker. Nevertheless, as already emphasized, professional workers among children will function on a higher plane and will avoid occasional unfortunate blunders if they possess some appreciation of the great diversity of the problems and some inkling of the nature of the rarer types. Even a limited knowledge of special types, causative factors, and treatment techniques should be of interest and value to both the professional and lay workers who are concerned with the care, treatment, and education of deficient or deviating children and with the practical eugenic and euthenic problems they create in society. Of course, nothing less than a comprehensive view of the subject will prevent the formation of warped judgments among professional workers and enable the trail blazers to construct an adequate foundation for a science of the prevention and amelioration of mental inefficiency and for the increase of mental efficiency.

Methods of differentiating special types

Some of the special types can be easily recognized by an expert on mental and neurological defects from gross physical signs and stigmata, and even by any intelligent observer after

some familiarity with the characteristics of the several types. This applies to some, if not all, cases of mongolism, cretinism, microcephaly, dwarfism, gigantism, gargoylism, hydrocephalus, epilepsy, paralysis, Friedreich's ataxia, and the Fröhlich syndrome. Many others that cannot be readily differentiated by surface inspection require thorough physical examinations, including, in certain cases, neurological, psychological, psychiatric, endocrinological, biochemical, and blood tests, and genetic investigations. The accurate appraisal of the degree of mental deficiency requires the correct administration of a variety of standardized psychological tests, particularly those of a clinical nature. Reference was made to such tests in Chapters 2, 3, and 6.

Accuracy of classifications according to clinical type. Too many gaps still exist in our knowledge of some of the clinical types to justify the pretention that any classification that can be made at the present time is strictly scientific, or completely accurate, or the best possible classification. Improved research techniques are constantly bringing new insights and requiring the discarding or the revision of old postulates. Ultimate knowledge of many of the types here discussed is a long way in the future. Many current explanations must be held as provisional hypotheses. Although causal relationships are assumed to exist between the types here discussed under a given rubric—for example, disorders of the endocrine glands and of nerve metabolism—nevertheless, mistakes in classification doubtless exist because of imperfections in scientific knowledge. Moreover, a few types are included in certain groupings as a matter of convenience because of outward similarities, although the causal nexus may be entirely different. Thus mongolism is treated in connection with endocrine disorder although some authorities continue to be sceptical regarding its endocrine genesis. Pygmies and midgets are considered in connection with dwarfs of the endocrine type (the Lorain-Levi and achondroplastic dwarfs) as a matter of convenience: they are not endocrinopathic disorders, so far as

is now known. The reader is cautioned to keep these reservations in mind to avoid making unjustifiable deductions.

Endocrinopathic disorders

Dysfunctioning of the endocrine or ductless glands and mental disorders. The body contains a network of interrelated and interdependent ductless glands, known as endocrine glands or glands of internal secretion, which secrete vital chemical substances directly into the lymph or blood. These substances, known as hormones or autocoids, regulate metabolism, tissue differentiation, mental and physical growth, sex activity, the general energy and efficiency of the circulatory, gastrointestinal, muscular, and nervous systems, and the individual's personality make-up. The endocrine glands include (1) the thyroid; (2) the parathyroids; (3) the pituitary; (4) the thymus; (5) the adrenals; (6) the islands of Langerhans (in the pancreas); (7) the gonads or sex glands; and possibly (8) the pineal. Each hormone exercises a specific influence on the body economy, but the glands also influence one another and the total effect depends upon this joint action. The health of each gland affects the functional efficiency of the other glands. The malfunctioning of one gland may impair the efficient action of one or more other glands. The glandular effects depend, in general, on whether a given gland is underfunctioning (*hypo-*) or overfunctioning (*hyper-*), on the amount of the deficient or excess discharge, and on the age of the person affected. All degrees of malfunctioning exist and the mental and physical effects produced vary all the way from barely discernible disturbances to the most profound psychosomatic disorders. Slight deviations are doubtless far more frequent than large ones, but may often go unrecognized. Many adjustment difficulties are caused by endocrine dysfunctioning, but the causal relationship may not be recognized as such. Serious organic or functional diseases or disturbances of the endocrine glands, known as endocrinopathies, are responsible for certain types of infantilism and

of mental deficiency, as well as for other mental and physical abnormalities.

Infantilism. The term infantilism has often been used without any very definite connotation. It has been broadly applied to the survival of infantile or childish traits into maturity, or (particularly in the field of mental hygiene) to the reversion or regression on the part of older children or adults to modes of feeling and thinking, attitudes, and behavior patterns that are characteristic of children or primitive people and should have been outgrown and abandoned at given stages of maturity. In general, infantilism connotes

EMOTIONAL IMMATURITY or childlike emotional survivals. Some persons who attain intellectual maturity or superiority may remain emotionally immature or childlike.

INTELLECTUAL RETARDATION, immaturity, or childishness. Although some of the victims may be intellectually normal or bright, many are intellectually backward or mentally deficient, depending upon the type and the degree of the glandular or other involvement. Some of those with fair degrees of intelligence may, nevertheless, be characterized by more or less intellectual childishness.

SEXUAL IMMATURITY, characteristic of certain types, especially glandular types, ranging from undeveloped genitalia and absence of sex impulse or amenorrhea to the lack of development or only partial development of secondary sexual traits, such as the failure of the growth of beard or pubic hair. In some adult endocrine disorders reversions to sexual infantilism occur.

BODILY ABNORMALITIES in certain types, particularly of endocrine origin, including dwarfed physical stature, the excessive accumulation of fat, and the disproportionate development of certain body parts, such as the feet, hands, trunk, head, or limbs.

The particular symptom complex of any individual depends on the type of infantilism and the age of onset. Many kinds of infantilism exist with varying mental and physical

characteristics. They may be grouped into three major classes.

TYPES. (1) "Idiopathic type" is a term designed to conceal ignorance of the real causation. In these cases no serious general or local disease or definite antecedent factor can be detected. Gilford's "ateleiosis,"⁸ a condition of continued youth or incomplete development (*a*, not; *teleios*, complete) may belong here, although some think the condition is caused by defective functioning of the anterior pituitary lobe. Ateleiosis is marked by a small jawbone, a childish facial appearance (facial juvenilism), small, slender limbs, short stature, a weak voice, conspicuous sex retardation, and very slow mental and physical development. Although the intelligence may have attained normality, it has been very slow in maturing. One reported case required 35 years to reach a five-year bodily stage. Some become prematurely senile (progeria, after Gilford) and may succumb at about twenty with all the signs of old age.

(2) Types caused by early disease or constitutional developmental defects, including diseases or disorders of: the kidneys (renal infantilism, Leonard Parsons), the pancreas (Bramwell's pancreatic infantilism), liver (hepatic infantilism, associated with hepatic cirrhosis), the lymph (lymphatic infantilism, from morbid growth or lymph tissue), the vascular system (angioplastic infantilism), and the intestines (intestinal, celiac, toxic, cachectic, or Herter's⁹ infantilism). These alleged varieties of infantilism require more careful study and discrimination than they have received thus far and will not be discussed in this book. Early syphilitic infection sometimes produces symptoms of psychic infantilism.

(3) Types produced by endocrine dysfunctioning, which may be caused by inherent or constitutional glandular defects

⁸ Gilford, Hastings, *Disorders of Postnatal Growth and Development*. London: Adlard & Co., 1911.

⁹ Herter, Christian A., *On Infantilism from Chronic Intestinal Infection*. New York: The Macmillan Company, 1908. (Cachectic: *kakos*, ill; *exis*, habit.)

or by lesions produced by accidents, abnormal growth (for instance, by tumors), infections, or otherwise. Toxins are supposed to be very injurious to the organism in the early weeks of fetal life before the functions of the adrenal glands have been established. These glands secrete lipoids, fatlike substances that are supposed to be needed for the development of the nervous system and for its protection from toxins (Frederick W. Mott). The most important glands from the standpoint of infantilism and mental defect are the thyroid and, perhaps, the pituitary. The relation of the parathyroids, pineal, adrenal, and the thymus glands to infantilism and mental defect is still shrouded in considerable obscurity. Our discussion of infantilism associated with endocrine disorders will include cretinism and mongolism and, more briefly, certain disorders of the pituitary, adrenal, thymus, and pineal glands.

Infantilism is not a general characteristic of the whole group of the mentally deficient, but it is a prominent feature in cretinism, mongolism, Fröhlich's syndrome (pituitary infantilism), and in some microcephalics, spastic diplegics, and juvenile syphilitics.

Chapter 10

CRETINISM (MYXEDEMATOUS INFANTILISM)

Cretinism is caused by the congenital lack of development (aplasia) or early atrophy or functional inactivity of the thyroid gland. The thyroid gland, which weighs about an ounce and is composed of two lobes that straddle the trachea (wind-pipe) at the level of the larynx, secretes a powerful hormone known as thyroxine, of which iodine is an essential constituent. Excessive thyroid secretion accelerates the vital processes and the rate of metabolism, increases the temperature, decreases weight, and produces insomnia, restlessness, hyperactivity, nervous irritability, and sometimes exophthalmic goitre (Graves's disease). On the other hand, lack of secretion (athyroidism) or seriously deficient secretion (hypothyroidism) at birth or early life produces a low grade of mental defectiveness known as cretinism. The condition is known as infantile, childhood, and adult myxedema according to when it originates.¹ Hypothyroidism is characterized by a slowing of the rate of metabolism (as shown by the basal metabolism test) and of the vital processes, subnormal temperature, mental dullness, sluggishness of movement, and dropsy-like swellings or obesity.

Endemic and sporadic forms

Cretinism occurs in two varieties: (1) the endemic or goitrous form, which occurs in various goitrous locations.

¹ For a detailed discussion of cretinism consult Benda, C. E., *Mongolism and Cretinism*. New York: Grune & Stratton, 1946. A brief delineation of adult myxedema is found in Wallin, *Personality Maladjustments and Mental Hygiene* (2d edition), 1949.

such as the Alpine valleys of Switzerland and Italy, the Pyrenees, the Andes and Rocky Mountains in America, the Himalayas in India, in Burma, in China, and elsewhere. It occurs in areas, whether of high or low altitude, in which a shortage of iodine exists. Normally from one-third to one-half of the body's iodine is contained in the thyroid gland. In the iodine deficiency form, the lack of thyroid secretion affects both the mother and the child.

(2) The sporadic form, the prototype, occurs in isolated instances in children with inactive thyroids who are the offspring of parents with functioning thyroids, at least in many cases. Although there are certain differences between the two types (for instance, the thyroid gland may be enlarged in the endemic form), the essential basis is the same, namely, functional inactivity of the thyroid gland.

In the sporadic form, which is the most prevalent in the United States, the child may appear normal at birth and may continue to appear so as long as he is nursing, provided the mother's thyroid is functioning properly. In such cases after the child ceases to nurse, the cretin symptoms become gradually apparent. Some cases, however, can be diagnosed almost at birth by the specialist, especially with the aid of X-rays of the ossification centers (for example, of the skull). Among the suspicious signs in the infant are loose, wrinkled, pale, or yellowish skin, especially over the forehead, difficulty in nursing, swellings in various parts of the body, apathy, sleepiness, slowness of movements, deficient temperature, open anterior fontanelle, gradual enlargement of the tongue, and delayed attempts to sit, walk, or talk. Completely developed intrauterine cretinism often results in a stillbirth.

The distinction between the congenital and infantile form, which may develop very early in postnatal existence, is not always easy. Some of the infantile and childhood forms may be due to the same cause as endemic goitre.² An inactive goitre might assist in the diagnosis of such cases.

² Hurxthal, Lewis M., "Cretinism," *The Medical Clinics of North America*, January, 1918, 122-139.

Physical characteristics of the fully developed cretin

(See Fig. 7.) Because of better diagnostic facilities and the institution of early treatment, good specimens of cretinism are now less frequent in the United States than was formerly the case. Moreover, not all physical and mental characteristics are equally prominent in all cases, indeed not all are present in all cases, even in the untreated ones, because the degree of functional activity of the gland may vary from complete impotency to imperceptible degrees of hypofunctioning.³ The terms cretinoid and demicretin are sometimes applied to the slighter degrees of cretinism. The salient bodily characteristics of the fully developed form include:

Thyroid: functionless, the primary seat of the disorder. The thyroid is often impalpable in the sporadic cases (athyreosis) and merely consists of a few follicles of the thyroid tissue. In the endemic type the thyroid, although functionless, may be greatly enlarged, sometimes three or four times the normal size, probably as the result of compensatory overactivity of the pituitary gland, which may be trying to make up for the lack of thyroid secretion.

General body appearance: bloated (edematous) from the accumulation of a semi-fluid under the skin; baggy folds; redundant skin; protuberant, pendulous abdomen; umbilical hernia common. Cretins are voracious eaters.

Skin: pallid or sallow; dry, scaly, and baggy; fatty tumors above the clavicles or in the axillae.

Head: heavy, large, long (dolichocephalic) with normal or low cephalic index, broad behind, flat on top; fontanelle late in closing.

Neck: usually thick and short.

Brain pathology: surface often simply convoluted; numer-

³It is important to emphasize at the very outset of the clinical characterizations of the different special types that not all of the mental and physical features enumerated are found in all cases. This broad generalization applies to every clinical type. The characterizations supplied are, in general, the most outstanding or the most prevalent ones.

ical deficiency and nondevelopment (hypoplasia) of pyramidal cells; sometimes sclerotic areas, hemiatrophy, and hydrocephaly.

Hair: harsh, coarse, scanty; often black.

Face: broad, lips thick and often protuberant; lower lip often everted; ears, large and flexible; mouth often open, driveling at times; tongue thick and sometimes protuberant; eyes widely separated; eyelids puffy with narrow palpebral fissure; forehead usually wrinkled; features coarse, expression dull, apathetic, immobile, and unobservant.

Nose: broad and flat with depressed bridge and flexible tip.

Hands: broad, thick; fingers short; finger tips squarish; skin often flabby and wrinkled.

Feet: squat; gait slow and waddling.

Stature: dwarfish (commonly about three to four feet); thickset body; legs very short, thick, and bowed; hands and feet short and stumpy. Ossification of bones retarded. A child of eight or ten may appear like an infant of two or three, as in Fig. 7. The stunted cretin growth contrasts with that of the achondroplastic dwarf whose trunk is fairly normal in length while the legs and arms are very short.

External genitalia: often infantile; puberty often delayed; usually sterile.

Hearing: often impaired.

Pituitary gland: often enlarged.

Temperature: subnormal.

Chronic constipation.

ILLUSTRATIVE CASES. A. C. (Fig. 7) is a sporadic cretin of idiot grade, aged 10.5 years when photographed, who, at the age of ten, measured less than a year by the Binet scale. His skin is rough, coarse, sallow, with purplish mottling, eczema on ears and genitals, scalp dry and scaly, hair sparse, eyes puffy, abdomen prominent (26 inches), arms and legs short, feet flat, legs held in flexed position. Has umbilical hernia. Under administration of thyroid extract, hair has grown thick, skin smooth and soft, and eczema disappeared. Walked with support at two, unable to walk, talk, and stand alone or feed himself, but wheels himself

about day-room in wheel chair. Very slow in movements. Can string beads and put pegs in peg board at random. Notices toys and plays with them. Ordinarily good-natured, but has bad temper spells, and cries readily when crossed.

The following demi-cretin in Delaware, whose career could be followed for several years, achieved greater progress than any other treated cretin examined in any of the writer's clinics.

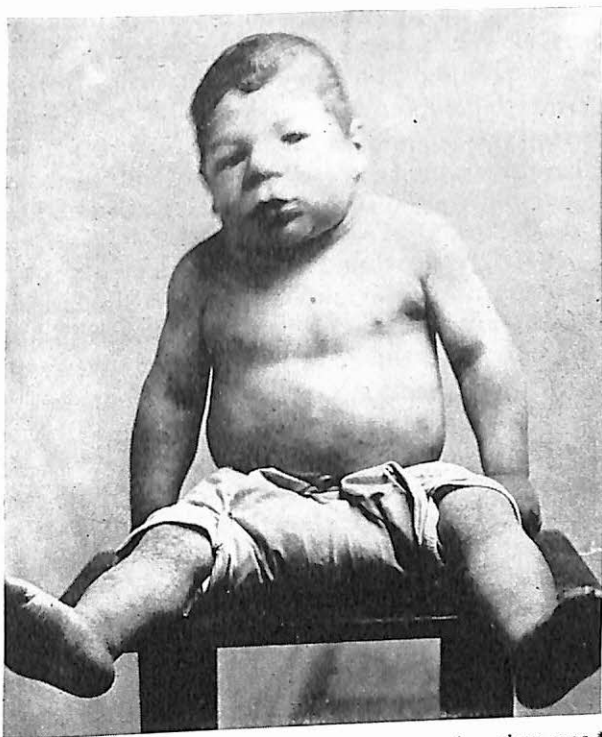


FIG. 7. Sporadic cretin, age 10.5, Binet age less than one.*

Pearl (born on May 14, 1930) did not enter a State Board school until she was 8.3. She was unable to do any of the work of the first grade, her speech and muscular coordination were very poor, she was dwarfish in stature, had a protuberant abdomen, was timid, excitable, very sensitive, and easily provoked to crying. She made a score of 21 on the Detroit First Grade In-

* Wallin, J. E. W., *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1939.

telligence Test, equivalent to an intelligence age of 4.9, and was referred to the division for a psychological examination. At the age of 8.8 she had a Binet age of 4.7 (Form L), and an I. Q. of 53. She failed on practically all of the Wallin-Gilbert (now Cutsforth) tests. The history showed that Pearl did not take her first steps until she was two years old or speak in short phrases until she was over six. She had been placed under thyroid treatment (3 grains a day) at about seven and a half years of age because of hypothyroidism. At that time according to the mother she weighed 30 pounds and was 31 inches tall. At the time of the psychological examination, a little over a year later, she had grown 11 inches and gained 9 pounds. The thyroid treatment increased her mental alertness and improved her appetite. In May, 1941, at the age of eleven, she weighed 58 pounds, a gain of 28 pounds since the thyroid treatment began. This weight is about normal for a nine-year-old girl 50 inches tall. Her net height was 44.5 inches.

In May, 1941, after two and a half years in the opportunity class, she had no noticeable speech defect, although she talked very slowly. Her muscular coordination was still rather poor, and she did not reach the standards for her age group in the stunt chart of the physical education department, but she could jump a rope and participate in many of the recess activities. She had reached the second grade level in reading. In September of 1941 she almost made the second grade score in the Gates reading test. She did second grade arithmetic, wrote fairly well, and was very good in oral and written English. She planned a dramatization, selecting the story and characters and reading the directions. She was obviously pleased by her success. Her greatest deficiency was psychomotor retardation: she was slow in everything she did. It was difficult to prevent the other pupils from "babying" her because of her short stature and chubbiness. Consideration was given to returning Pearl to "the regular grades next year to see how she will react to a normal group."

Two years later at the age of thirteen she was "more mature mentally but still suffering from nervous reactions, is in the third grade and doing work on that level, and will be promoted in June to the next grade. The teacher looks for restoration in two years."⁴

⁴ State of Delaware, *Annual Report of the Department of Public Instruction, Division of Special Education and Mental Hygiene, 1940-41*, pp. 311 f.; 1942-43, p. 293.

In May, 1944, at the age of fourteen, she was in the fourth grade in a rural school but was rated by the teacher as doing only second grade work in reading, arithmetic, and other school subjects. She followed directions readily but displayed no leadership qualities. She played with younger children, got along very well with them and, although better adjusted than a year ago, was still very nervous. She had not grown any physically.

In May, 1946, at the age of sixteen, she was in the fifth grade, responding very happily when complimented, and assisting in the housework at home. She had a Stanford-Binet Form L age of 8.4, I. Q. 56 (as compared with 4.7 and 53 in 1938 at the age of 8.8), an Arthur Performance age of 9.1, I. Q. 57; a grade III plus rating in reading, written language, arithmetic, and spelling on the Wallin-Gilbert (Cutsforth) Individual Attainment Scale (no norms above grade III are available), as compared with almost complete failure in 1938. She scored grade 3.6 in the Woody-McCall Mixed Fundamentals, 4— in Monroe's Standardized Reading Test, and 4.5 in Gray's Standardized Oral Reading Paragraphs. Her educational achievement level is about on a par with her intelligence age. Her height was 44.5 inches (about equivalent to a seven-year-old girl) as compared with about 31 inches at the age of about seven and the same height at the age of eleven. She continued to take three grains of thyroid extract a day, except for a period of almost three months in 1946. Her weight during this interval increased from 102 to 110 pounds, the abdomen became protuberant, she grew too large for her clothes, and became irritable. It is probable that this child would have become mentally normal had the treatment been started during the first year of life.

Mental and educational characteristics of untreated cretins

The typical cretin who has remained untreated or who has not profited from treatment is apathetic, listless, heavy, unobservant, but good-natured, docile, placid, and rather imperturbable. His speech is greatly delayed, thick, low-pitched, and indistinct. Although inclined to be somewhat timorous, resentful of interference, and restless occasionally, he is easily managed and harmless. It may have been because of the inoffensiveness and guilelessness of these simple innocents that

the French called them cretins, from *chrétien*, the French word for Christian. The intelligence level is much lower than that of the general run of primary cases. Marked untreated cases are usually speechless idiots. Most untreated cretins make little educational progress, although they can be trained to acquire useful habits of response and to conform to a simple household routine. No cretin should, however, be denied entrance to a public school merely because he is a cretin, as has been too frequently done. With early thyroid medication some can do acceptable work in a special class and a few can function in the regular grades. The average Binet age of ten of my St. Louis and Ohio cases, some of whom had received thyroid treatment, was 4.8 (average of the ratings from the 1908, 1911, and Stanford scales) and the average I. Q. 43, with a variation in Binet age from 2 to 7 and in I. Q. from 24 to 68.⁵ Seventy per cent were diagnosed as imbeciles, 20 per cent as potential morons (who, though rating as imbeciles at the time, would probably eventually advance to the level of morons), and 10 per cent as morons. Of four cretinoids, one was diagnosed as a moron, two as potential morons, and one as an imbecile. One had a Binet age of 2 and three of 7. One had an I. Q. of 38, one of 59, and one of 68.⁶ Nine is reported to be the highest intelligence age among cretins.

Causes of cretinism

Among the numerous hypotheses advanced in explanation of cretinism, note may be made of the following:

(1) One of the earliest conjectures, now largely defunct, was that endemic cretinism was caused by lime or some organ-

⁵ It was impossible to evoke any responses whatever from one of the ten, an eight-year-old girl, weight 112 pounds, who would not even make warding-off movements toward objects thrown at her.

⁶ Wallin, "The Diagnostic Findings." Two more demicretins were found among later Ohio cases, boys, Binet ages 5-8 and 4-2, I. Q.'s 77 and 56, diagnosed as morons. Curiously, only one cretin came to my attention from among over 10,000 examinees in my three Delaware clinics, but I had clinical contact with only a few of these cases because of excessive administrative and supervisory activities.

ism in the drinking water or miasma (noxious vapors) in the air of the valleys affected.

(2) The observation that families moving into goitrous districts were apt to develop goitre in the first generation and cretinism in the second and succeeding generations has suggested the theory that cretinism represents the degenerative end-product of endemic goitre. The inference is that when goitre has existed in our goitre belts in the United States as long as it has in Switzerland or India, we will have an equally large proportion of endemic cretins. It is supposed that the lack of iodine in the water and soil is the cause, or at least a contributing cause, of the inactive goitres, and thus of endemic cretinism as well, although certain geographical areas of endemic cretinism exist that are not far from the ocean (as in England).

The theory is also held that various "goitrogenic" substances (infective or toxic substances) might prevent the liberation of iodine within the thyroid or its absorption and thus produce a functionless or toxic goitre. Iodine deficiency in the mother produces iodine deficiency in the fetus and also, as a consequence thereof, deficiency in the vital thyroid hormone. The thyroid hormone is indispensable for the normal development of the fetus, particularly during the later months of its intrauterine existence. Some goitrous mothers are unable to secrete enough iodine to form an active thyroid in the infant.

William S. Halsted in 1896 produced experimentally signs of cretinism in puppies by removing large parts of the thyroid in the pregnant mothers and withholding iodine from the diet. Later litters showed no signs of cretinism when the mother had been fed iodine.

(3) Andre Crotti maintains that goitre is a fungus growth produced by the same spores that generate mold in bread and mushrooms. In support of his theory he cites the fact that he found a specific fungus (minute golden bodies) in the goitre of human beings, in the water of goitre areas, and in

cabbage. He produced goitre in dogs and rabbits by feeding them cultures from the same fungus. Iodine fed to the rabbits did not prevent the growth of the goitre.

(4) John T. King, on the other hand, maintains that exophthalmic goitre, characterized by "pop-eyes," is intimately connected with chronic tonsillitis; the lymph channels of the thyroid and the lymph glands of the neck (including the tonsils) are closely interconnected.

(5) Inflammation of the thyroid gland of the embryo from congenital syphilis has been suggested as a possibility. Other toxic agencies might also act on the thyroid through the mother's blood.

(6) Although many writers think that heredity plays an insignificant role in the causation of cretinism, John Thompson and Tredgold have found evidence of neuropathic inheritance in certain cases, leading Tredgold to postulate an hereditary type, in which hypothyroidism is a superadded complication. This type does not improve mentally from thyroid treatment.⁷ Penrose also thinks that "hereditary influences are the determining factor in the cases that do not respond mentally to treatment." He believes there may be an "hereditary deficiency of the thyroid to utilize iodine."⁸

(7) Severe emotional disturbances in the mother, particularly during the period of gestation, have been postulated, the assumption being that such disturbances might inhibit the thyroid secretion or injure the gland.

All authorities agree that cretinism is produced by excessive hypothyroidism from early life, but the basic cause of the inactivity of the gland has not been definitely established.

Treatment of cretinism

The recognized treatment, since it was introduced in 1892 by George R. Murray and Frantz Howitz, consists in the administration under medical supervision of varying daily doses (from $1\frac{1}{2}$ grain to 4 or 5 grains) of extract from the

⁷ Tredgold, *Mental Deficiency*, p. 288.

⁸ Penrose, *Mental Defect*, p. 130.

thyroid gland of the sheep. The proper dosage varies with the age and condition of the subject. The essential hormone, thyroxine, can be substituted in greatly reduced dosage. Overdoses may produce restlessness, hyperactivity, violence, and insomnia. The effectiveness of the thyroid therapy depends on the age of the affected person when the treatment was inaugurated, and the severity and type of the affection. Early treatment may almost completely remove the physical stigmata. The child loses his excess weight, he increases rapidly in height—one child is reported to have grown three inches in six months—the skin becomes moist and loses its sallowness and puffiness, the hair becomes softer, and the temperature increases. Ordinarily little change occurs when the treatment has been delayed until adulthood.

Mentally the child becomes more alert, interested, observant, vivacious, and responsive. He begins to develop some initiative and to participate in the activities of other children. When the treatment is begun during the first few months or possibly the first year, he may be restored to mental normality. But this does not always happen. Although a primary type of mental defective with hypothyroidism as an added handicap may become superficially brighter, even to the point where the nonexpert may believe he has been restored to normalcy, nevertheless, he continues mentally defective throughout life, although the physical stigmata may have largely vanished. Lapage's statement is in accord with the author's experience: "the average case never gets beyond a certain point, and is feeble-minded for the rest of his days."⁹

It is, of course, possible that most of those receiving early treatment have not reached my clinics and that most of the few who have, have been primary cases of mental defect. Gesell, Amatruda, and Culotta¹⁰ report that four of six female cretins receiving treatment from one year to ten years were

⁹ Lapage, C. P., *Feeble-mindedness in Children of School Age* (2d edition), p. 100.

¹⁰ Gesell, A. L., Amatruda, C. S., and Culotta, C. S., "Effect of Thyroid Therapy on the Mental and Physical Growth of Cretinous Infants," *American Journal of Diseases of Children*, 1936, 52:1117-1138.

brought from the defective to the normal level. They contend that the final outcome is dependent less on the "age of the child or his maturity status at the time of the diagnosis" than on his "residual physiologic capacity and the latent growth potency of his neuroendocrine system. Thyroid therapy cannot bring about normality if there has been a fundamental impairment of that system, hereditary or developmental." Most authorities would probably regard the psychological prognosis as fairly good in the case of cretinism beginning after birth during early childhood, if the treatment is begun early and if the cretin possesses good constitutional potentials. The combinations of thyroid, pituitary, suprarenal, pineal, and other extracts have proved disappointing with cretins of limited intellectual endowment. To obtain satisfactory results with promising cases it is imperative to start the treatment early and to continue it indefinitely. Weak doses of thyroid extract are often highly efficacious in the case of persons affected by mild degrees of hypothyroidism. Incidentally, iodine is effective in the treatment of simple goitre.

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Instances of restoration to mental normality through thyroid grafting appear in—

Veronoff, Serge, *From Cretin to Genius*. New York: Alliance Book Corporation, 1941.

The restoration of a congenital cretin, who was fed thyroid extract as early as the seventh week, to physical and mental normality is shown in four photographs in Kleiner, Israel S., *Human Biochemistry*. St. Louis: The C. V. Mosby Company, 1945, p. 494. (Chaps. 12 and 23 contain brief discussions of vitamins and hormones.)

Prevalence of cretinism

The following percentages have been reported, in some cases without distinction as to type.

In England: George F. Still—2.8 per cent among hospital cases. Lapage—0.7 per cent of 784 children admitted to

special classes in Manchester and 2 per cent of 120 rejected as too defective.¹¹ Two and one-tenth per cent, apparently of examinees for special classes in Essex. Penrose—from 3 per cent to 5 per cent of all institutional cases present “a fairly marked degree of subthyroidism.”¹²

In Switzerland: Hans Eggenberger—1.25 per thousand (endemic form).

In Hungary on the Island of Schutt: Endvay—1.3 per cent (endemic form).

In the United States: Halperin—2.3 per cent of consecutive admissions from Northern Ohio to institutions for the mentally defective. Yannet—0.53 per cent of consecutive institutional cases in Connecticut. Wallin—0.3 per cent of examinees in the St. Louis psychoeducational clinic, constituting 0.8 per cent of all the feeble-minded and 2.0 per cent of the imbeciles. And 0.1 per cent of 3,960 examinees in the psychoeducational clinic of the Bureau of Special Education of Miami University in Ohio (all sporadic cases).¹³ David C. Kimball and Harvey G. Marinos (according to Arnold S. Jackson)—8.2 per cent of 3,548 mentally subnormal children in Detroit. The latter estimate probably includes many cases of demi-cretinism and of mild hypothyroidism.

Penrose's observation that the condition, whether mild or severe, is much more frequently seen in females than in males applies to my own limited array.¹⁴

¹¹ Lapage, *Feeble-mindedness in Children of School Age* (2d edition), p. 56.

¹² Penrose, *A Clinical and Genetic Study*, p. 129.

¹³ Wallin, *The Education of Handicapped Children*, p. 346; “The Diagnostic Findings,” pp. 182 f.

¹⁴ Penrose, *Mental Defect*, p. 130.

Chapter 11

MONGOLISM

A challenging morbus incognita. Mongolism is a form of infantilism that superficially resembles cretinism, with which it is often confused in spite of clearly defined differences. It is one of the most interesting, curious, and inexplicable of the development anomalies of early life. Conflict of opinion has raged unabated for many decades regarding its true cause. Like epilepsy, it has remained a *morbus incognita* until within recent time. The recent investigations and extensive data amassed by Benda¹ appear to have brought some order out of the existing chaos and to justify the theory that mongolism is a major congenital pluriglandular defect whose primary locus is in the anterior pituitary lobe, due to a lack of development (hypoplasia) of certain microscopic cells or chemical elements. The name mongol was applied to the condition in 1866 by J. Langdon Down because of certain superficial facial resemblances to the members of the Mongolian race, particularly the obliquity of the eyebrows and of the fissure between the eyelids (palpebral fissure). These defectives are referred to indiscriminately as mongols, mongolians, mongoloids (which a few writers, including the author, limit to the slighter grades), and kalmucks. The term "congenital acromicria" (*akros*, extremity or end; *mikros*, small) has been applied to the condition (by Hans Schüller, W. Clift, and Benda) as an antonym of the acquired defect

¹ *Mongolism and Cretinism*, p. 6.

acromegaly (*megala*, large), in which the bones of the hands, feet, and face are enlarged.

Incidence

The mongols are far more numerous than the cretins. The ratio of mongols among institutional cases of mental defectives, children's hospital patients, and enrollees in, or candidates for, public school special classes for mental deficients is shown by the following figures.

In England: Langdon Down—over 10 per cent in hospitals and at Earlswood. Thomas N. Brushfield—8.4 per cent of 2,090 admissions of mentally defective children of two to ten years to Fountain Hospital. Lapage—0.5 per cent of 784 admitted to special classes in Manchester. Six per cent of 120 rejected as low defectives. Potts—"nearly 5 per cent of mentally defective children, in greater or less degree."² Dwight W. Hunter—not much over 6 to 8 per cent of mental defectives (the number is reduced by early deaths and the tendency to outgrow the mongolian condition). George F. Still—22 per cent of 350 cases of hospital imbeciles. John Thompson—10 to 15 per cent at least of mental defectives recognized as such in infancy. Tredgold—40 to 50 per cent of those diagnosed as mentally defective during the first year of life, and about 5 per cent of the total number of aments.

In Scandinavia: Sakari Lahdensuau—0.47 per cent of 8,517 examinees in the University Children's Hospital in Finland. H. Hellsten—0.63 per cent of 5,900 admissions to a children's hospital in Sweden, or 28.2 per cent of all diagnosed as defective.³

In the United States: Martin W. Barr—10 per cent of all idiots. Halperin—8.2 per cent of consecutive institutional

² Shuttleworth, George E., and Potts, William A., *Mentally Deficient Children, Their Treatment and Training*. London: H. K. Lewis & Co., Ltd., 1922, p. 61.

³ Jervis, George A., "Recent Progress in the Study of Mental Deficiency Mongolism," *American Journal of Mental Deficiency*, April, 1942, 467-481.

admissions. Wallin—0.86 per cent of 2,774 consecutive St. Louis psychoeducational examinees, being 2.3 per cent of those classified as feeble-minded and 5.8 per cent of the imbeciles (inclusive of five mongoloids); and 0.65 per cent of 3,960 psychoeducational clinic cases in Ohio (inclusive of three mongoloids). Bleyer—0.3 per cent of almost 50,000 children from two weeks to fourteen years of age seen in an out-patient department of the St. Louis Children's Hospital. The proportion of mongols among the mentally deficient children was 19.4 per cent. His findings yield an estimate of at least 28,000 mongols in the United States.⁴ Beidleman—0.5 per cent of the general population in children's hospitals (the mean of many studies).⁵

For the general population, Penrose reports almost 200 in a population of two million in the Eastern Counties of England, or one in 10,000; and Van der Scheer found 500 among eight million people in Holland, or one in 16,000, with no sex difference. These may be underestimates because of the unreported mongols who suffered early extinction.

On the basis of a summary of seven studies involving 3,097 mongols, Jervis found that 56.3 per cent were males and 43.7 per cent were females.⁶ Beidleman found a ratio of 145 males to 100 females in institutions, based on four studies.⁷ The excess of males in institutions has been attributed, rightly or wrongly, to the greater willingness of parents to part with their boy mongols and the greater number of male behavior cases. There was no marked sex difference among my St. Louis extra-institutional examinees, but a distinct preponderance of boys among the Ohio cases.⁸

⁴ *Ibid.*, p. 467.

⁵ Beidleman, Barkley, "Mongolism: A Selective Review," *American Journal of Mental Deficiency*, July, 1945, 35-53 (97 titles).

⁶ Jervis, *op. cit.*, p. 468.

⁷ Beidleman, *op. cit.*, p. 36.

⁸ Wallin, *The Education of Handicapped Children*, p. 346; "Mongolism Among School Children," *American Journal of Orthopsychiatry*, 1944, 104, 107.

Physical characteristics

A bewildering array of physical stigmata is characteristic of mongolism, but not all anomalies are found in every mongol. A further complication is that some mongol peculiarities are found in other types of mental defectives and even in some mentally sound persons. Instead of a dichotomous distribution one finds, rather, an imperceptible gradation in the intensity and number of these defects from normal persons down through slight mongoloids to profound mongols. In a study of 50 mongols and 350 unselected mental defectives, Penrose found that almost 75 per cent of the mongols had three or more of the mongol traits as compared with only six cases among the unselected defectives. He concluded that any defective with "four or more of these characters is almost certainly a mongol." The author has for many years considered that three or more of the anomalies are pathognomonic of mongolism. The most prevalent characteristics found by Penrose among the mongol as compared with the non-mongol defectives are: a transversely fissured tongue, 74 per cent versus 7.2 per cent; an epicanthic fold on either eye at the inner angle or canthus of the eyelids, 52 per cent versus 3.4 per cent; a transverse palmar line on either hand, 44 per cent versus 3.8 per cent. These rankings differ considerably from Myra Kuenzel's most "differentiating" traits based on a study of 31 mongols in comparison with 31 non-mongol mental defectives of the same sex, life age, and intelligence age. The four most important traits in descending order were: almond-shaped eyes, flattened occiput, depressed nasal bridge, and pointed tongue.⁹

Although all mongols look alike superficially, the mongol characters are more or less inconstant with individuals, and some—for instance, the epicanthic folds and the shape of the

⁹ Kuenzel, Myra W., "A Survey of Mongolian Traits," *Training School Bulletin*, 1929, 49-60.

head—tend to attenuate or change with age, so that it may be difficult to recognize some older persons as mongols. Although mongolism is a congenital defect and the physical peculiarities are apparent at birth to the discerning eye,¹⁰ a correct diagnosis of the condition in many cases is not made for many months or years. The chief abnormalities affect the head, eyes, nose, tongue, hands, feet, and stature.

Skull: small (usually with a maximum circumference of not over 19 inches at maturity, equal to about a four-year-old), roundish and tending to become brachycephalic (broad headed, with a cephalic index of over 80); absence of bossing and the occipital protuberance, but late closing of the large anterior fontanelle and the sutures.

Face: small, broad, flat, plump, moon-shaped, with a bland, childish expression; florid cheeks, complexion often mottled; without prominent cheek bones; nonreceding forehead, usually smooth.

Neck: short and thick.

Hair: tends to become coarse, wiry, sparse; often mouse-colored; often downy growths on neck, cheeks, and forehead.

Brain pathology: evidences of pressure and deficiency of oxygenation—gross distortions and phenomena of degeneration; small, simple convolutions; thin cortex; many imperfect cells; reduced pyramidal cells; lack of association tracts; demyelination; small cerebrum, pons, medulla, and cerebellum.

Mouth: small and round; lips sometimes thickened and fissured vertically; upper short, lower often everted; small mouth cavity; palate often high and narrow.

Nose: squat, small, with sunken bridge.

Eyes: commonly small, almond-shaped and slanting; eyelids

¹⁰ Ninety per cent can be recognized on the day of birth, according to Aldrich, who makes a plea for institutional commitment during babyhood, to save the family from eventual bitter disappointment and "almost hopeless entanglement of emotional ties to the mongol," by the mother. Aldrich, E. Anderson, "Preventive Medicine and Mongolism," *American Journal of Mental Deficiency*, October, 1947, 127-129.

puffy or thin, with narrow palpebral fissures slanting downward and inward; scant eyelashes; epicanthic fold (a skin fold at the medial angle), which disappears at about the age of twelve; small orbit holes; frequent convergent strabismus, nystagmus (rapid, involuntary oscillations of the eyeballs), cataracts, ptosis (drooping of the upper eyelids), and conjunctivitis; obliquity of the eyebrows.

Tongue: often protrudes at birth; may be protruded and retracted constantly; large (at least in relation to size of oral cavity) and soggy, or narrow and pointed; coarse papillae (circumvallate papillae) may appear between the third and ninth month or later; transverse fissures may appear by the fourth year (sometimes ascribed to tongue sucking).

Jaws: lower tends to protrude (prognatism); upper small.

Ears: often crumpled, protruding, and small; sometimes large and flapping; sometimes dissimilar; Darwinian tubercle sometimes prominent; lobule deficient.

Skin: tends to become coarse, dry, scaly, rough, and often downy, depending on the degree of hypothyroidism.

Abdomen: large and distended; umbilical hernia frequent.

Genitalia: almost rudimentary; menarche late or none (in 50 per cent); lack of libido.

Heart: various congenital anomalies frequent; poor circulation and blue and cold extremities.

Stature: dwarfish, the greatest growth retardation occurring during the first three years and affecting particularly the distal ends of the long bones; retarded bone ossification; a child of eight or nine looks about like a child of four or five, an adult male rarely exceeds five feet; straight, normal trunk with short, thin limbs; laxity of the ligaments, hyperextension of the joints, with unusual mobility of the wrists, fingers, hips, and ankles; tends to sit with legs crossed, tailor-fashion.

Feet: short, broad, round, clumsy, flat, with wide cleft between the big and second toes and a deep furrow from the cleft; occasionally supernumerary toes and syndactyly.

Hands: flabby, broad, clumsy, with short, thick, spread-out

fingers, the thumb and little finger especially short; inward curvature of the latter frequent; fingers often taper; wrinkled skin; transverse palmar crease frequent.

Thyroid: often deficient.

Strength: weak, delicate, lack of muscular tone; poor circulation; heart lesions common; subject to fainting spells, susceptible to respiratory infections, especially to tuberculosis and pneumonia (many now rescued by sulfapyridine); about 75 per cent die from infectious diseases; epilepsy very infrequent.

Mortality rate: the average age at death of Pearce and Rankin's 50 cases, 14.5 and of Kate Brousseau's 165 cases, 12.5; according to Penrose the average mortality rate for cases of all ages in institutions is about six times the average of all other defectives and nine times that for the general population; the mortality rate was about 57 per 1,000 for mongols as compared with 15 per 1,000 for non-mongolian defectives, based on a study of about 7,000 Illinois mental defectives.¹¹ Nevertheless, a few reach or surpass the half century mark.

Psychological and educational characteristics

A considerable proportion of mongols are lively, observant, and superficially bright. They are good-natured, easily amused, and exhibit a smiling countenance. They are affectionate, amiable, docile, and cheerful when intelligently handled, but are subject to stubborn spells when not tactfully treated. A certain proportion of them are shy, reticent, apathetic, and negativistic, and some are restless, noisy, uncontrollable, mischievous, destructive, and subject to bouts of temper or excitement. Henry Rollin¹² found behavior disorders of various kinds in 60.2 per cent of 73 "proved" mon-

¹¹ Leibowitz, Albert, and Yannet, Herman, "The Production of Humoral Antibodies by the Mongolian," *American Journal of Mental Deficiency*, 1942, 304-309.

¹² Rollin, Henry R., "Personality in Mongolism with Special Reference to the Incidence of Catatonic Psychosis," *American Journal of Mental Deficiency*, October, 1946, 219-237.

gols (who exhibited four or more of the mongol stigmata), ages eight to forty-eight. The behavior disorders of many cases apparently stemmed from a psychotic background. He classified 23.3 per cent as cases of mental disease of the "primitive catatonic" dementia praecox type (see Chapter 4).

Many mongols are imitative and show marked aptitude for mimicry, but they are lacking in initiative. Although often exhibiting a striking fondness for music and possessing a fairly good sense of rhythm, they make poor singers and clumsy dancers. Mongols as a class are very slow in learning to sit, creep, stand, walk, and talk. First steps may not be taken until the child is from three to six years old. His speech is guttural, harsh, and indistinct, and some never learn to speak. He is notably deficient in sensory discrimination and motor coordination.

Goddard, on the basis of 11 institutional cases, affirms that it is "a remarkable fact that the mentality of mongolian imbeciles is almost always that of a four-year-old child,"¹³ and Benda reports that nearly all of his older cases had a mental level of between three and four.¹⁴ Of Kuenzel's 31 cases, over half were classified as imbeciles, nine had mentalities of one and two, and the highest Binet age was 7.9.¹⁵ Thirty-eight per cent of Brousseau and Brainerd's 206 mongols were diagnosed as idiots, 61 per cent as imbeciles, and 1 per cent as morons.¹⁶ Rollin classified 57 per cent of his cases from the Catherham Hospital in England as idiots (I. Q.'s below 20, Form M, Stanford-Binet), 30 per cent as low grade imbeciles (I. Q.'s from 20 to 30), and 11 per cent as high grade imbeciles (I. Q.'s from 30 to 40). The highest I. Q. was 52.¹⁷ Penrose reported age seven as the "upper limit of their intelli-

¹³ Goddard, *Feeble-Mindedness, Its Causes and Consequences*, p. 453.

¹⁴ Benda, "Further Clinical and Pathological Studies of Mongolism," *Journal of Psycho-Asthenics*, 1939, 1:47.

¹⁵ Kuenzel, "A Survey of Mongolian Traits."

¹⁶ Brousseau, Kate, and Brainerd, H. G., *Mongolism: A Study of the Physical and Mental Characteristics of Mongolian Imbeciles*. Baltimore: The Williams & Wilkins Company, 1928.

¹⁷ Rollin, "Personality in Mongolism," p. 228.

gence." The author's St. Louis cases varied from Binet age —2 to 7-8, with a mean of 4-0, and the corresponding figures for the Ohio group were 2-6 to 6-8, with 3-6 as the mean (exclusive of those who could not be measured). The I. Q. range in the St. Louis group was from 19 to 63, with an average of 36.2; the corresponding I. Q.'s for the Ohio group were 19 to 53, and 37.1.¹⁸ The highest reported mean intelligence level emanates from the Bancroft School, a private school for defectives in New Jersey: 6-4 (from 2-9 to 10-8), with a mean I. Q. of 46.4 (from 24 to 71).¹⁹ It is not apparent from the data published whether this difference is due to "selective admission" into this private school, or to the inclusion of a large ratio of mongoloids, or some other factor. Three of the Bancroft cases, however, with Binet ages over seven years were twenty-two years of age and may have continued to improve after early adolescence. However, neither the Bancroft nor the writer's group confirms Penrose's conclusion that "the most likely range of intelligence for mongols" is the I. Q. range from 15 to 29. The modal I. Q. span for the St. Louis, Ohio, and Bancroft cases is 36 to 40. Ten per cent of 30 of the author's cases (mostly from St. Louis) were diagnosed as morons, 86.6 per cent as imbeciles, and 3.3 per cent as idiots. The average I. Q. of the St. Louis mongoloids (slight cases) is 46.4 and of the Ohio mongoloids, 46.6. Another array of 26 cases gave almost the same proportions. All the morons were classified as mongoloids as were several of the imbeciles also.

The educational prognosis is discouraging for most mongols. The majority make less progress than the average mental defective, and some progress even less than the average imbecile. Because of clumsy fingers and poor motor coordination, they are incapable of doing work that requires fine motor coordination.

¹⁸ Wallin, "Mongolism Among School Children."

¹⁹ Pototzky, Carl, and Grigg, Austin E., "A Revision of the Prognosis in Mongolism," *American Journal of Orthopsychiatry*, 1942, 503-510.

ILLUSTRATIVE CASES. D. M. (Fig. 8) is a mongol imbecile who was 12.4 years old when the photograph was taken, and who had a Binet age of three and an I. Q. of 21 at the age of 11.7. He can string beads, put pegs in a board, march in step, carry out one command, button clothes, imitate, but cannot tie a knot, and does not know colors or forms. He likes to pretend he is dusting and helping in the house.

Many, however, can be trained to do simple forms of art and craft work, including drawing and writing, to perform many simple, routine indoor and outdoor tasks, to conform to the ordinary rules and regulations of the home and the school, and to acquire many useful habits. The slighter grades, especially the mongoloids, may achieve perceptible success in literary work, but rarely in arithmetic. Although some can learn to count and do simple sums, most mongols are incompetent in the field of arithmetical comprehension. Curiously, a few possess considerable ability in reading, so far as concerns the mechanics of word calling (not the extraction of meanings), and reach a higher mechanical reading level than could be expected from their Binet level or I. Q. Many thus deceive their parents who may refuse to believe that they are mentally defective.

W. S. was one of the best readers, if not the best one, from the standpoint of the mechanics of reading, in one of my demonstration classes in Ohio, although his Binet level was several years lower than that of several other pupils in the class. At the age of 10-5 his Stanford-Binet was only 4-4 (with a basal age of three), I. Q. 42. His reading level (based on reading mechanics, not



FIG. 8. Mongolian imbecile, age 12.4. Binet age three at age of 11.7, I.Q. 21.*

* Wallin, J. E. W., *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1939.

comprehension), on the other hand, was high third grade at about the age of twelve, which was much above his absolute and relative Binet level. From the standpoint of his intelligence level he would classify as a near imbecile, but scarcely so from the standpoint of his reading achievement. The mother, an ex-public school teacher, who had set her heart upon educating him to become a lawyer, would not accept the diagnosis of mongolian defectiveness, withdrew him from the special class, and placed him in a private school. A pediatrician reported that he had been restored to near normality, but a retest showed little change in the I. Q. (the test results are not now available). He had become merely superficially brighter. When I last saw him a year or two later he had been withdrawn from the private school and was running the streets.

Only one or two mongols under my observation have done about as well in arithmetic as in reading. Richard, Stanford-Binet 6-4, I. Q. 48, at the age of 13-6, in the third grade in a rural school in Delaware, after five and a half years in school, rated grade III (the highest available norm) in the Wallin-Gilbert Individual Attainment Scale in reading, written language, and arithmetic, and grade II in spelling (phonetic words). The teacher rated him as third grade in reading and spelling, and second or third in arithmetic, with greatest aptitude in reading and adding, greatest deficiency in division, and greatest interest in farming. He was stubborn and shy before strangers.

Benda holds that mongols possess "dormant possibilities of improvement" and maintains that their education should not stop too early.²⁰ They should not be denied the privilege of receiving training in public school special classes, although transfer to appropriate residential institutions may be preferable, especially if there are other children in the home.

Four striking facts regarding mongolism

The first fact is that the mongols are born "unfinished" or "ill finished" through arrest of fetal development, in consequence of which Penrose has applied the term "fetalism" to the condition. Although Benda casts doubt on the value of

²⁰ *Mongolism and Cretinism*, p. 65.

the analogy,²¹ many of the mongol physical traits are characteristic of various stages of the normal fetus, such as the round head, the fetal proportions between the forehead and the face, the shape of the mandible, the puffy eyelids, the epicanthic folds (normal for the seven- or eight-month fetus), the short limbs, the shape of the hands, fingers, and toes, the laxity of the joints, and other traits. Someone has said that if you magnify the four- or five-month fetus 30 or 40 times you will get a full-blown mongol. Something that went wrong during embryonic life, dating possibly "not later than the eighth week of pregnancy" (Penrose), produced fetal arrest and mongolism.

Second, in the vast majority of cases only one mongol is born in a given family. The writer has encountered only one set of mongol siblings among some 19,000 examinees in seven psychoeducational clinics in six states. These siblings, examined in St. Louis, came from Arkansas. However, a number of writers have reported mongolian siblings, or mongolian twins, of whom one or both were mongols, or half sibs (the same mother), one of whom was a mongol, or a mongol and a cretin in the same family. One study lists four mongols in one generation; another three mongols among 14 siblings, with ten mongols in three generations.²² Such exceptions, however, are sufficiently rare to prove the rule.

Third, the discrepancies that exist in the many studies of the ordinal position of the birth of the mongol, the size of the families, and the age of the parents at the time of birth of the mongol are too numerous for brief summarization, but almost all findings are in agreement on the basic fact that the average age of the mothers at the time of the birth of the mongols is considerably higher than the corresponding age of mothers of non-mongolian defectives or of normal children. The average age of the mothers of 154 mongols in Holland was six

²¹ *Ibid.*, p. 15.

²² For further details see Tredgold, *Mental Deficiency*, pp. 209 f.; also references in Jervis, "Recent Progress in the Study of Mental Deficiency Mongolism," pp. 467-481.

years higher than that for mothers of 573 normal children (Van der Scheer). The corresponding difference for 215 mongols and 215 controls in Canada was 8.2 years (C. Roger Myers). Benda found that 40 per cent of 255 mothers were over forty years, but that only 3 per cent of the mothers of the primary or familial types of defectives were over forty. For my St. Louis cases the mothers averaged 5.4 years older than the mothers of non-mongol defectives and 6.1 years older than the mothers of all the clinic cases. Based on various studies of 282 mongols, Bleyer found the average age of the mothers of mongols to exceed the average maternal age of two million controls by more than ten years. The peak age of mothers of mongols was forty-one years compared with twenty-four years for mothers in the general population.²³ Nevertheless, however important the factor of advanced maternal age may be, advanced age in itself probably is not the cause of mongolism, for mongols are frequently born during the most favorable period for reproduction. Twenty-five per cent of the St. Louis mothers and 32 per cent of the Ohio mothers were in their twenties.²⁴

Fourth, a considerable proportion of the mothers of mongols have been in ill health, have suffered from thyroid deficiency, and have experienced miscarriages, long barren periods, and uterine hemorrhages. Many of the mongols were born prematurely. (Premature brains are more vulnerable than normal ones.)

Theories of causation of mongolism

The human mind has been extraordinarily fertile in propounding explanations of this mysterious developmental anomaly, most of which are highly conjectural and without

²³ Bleyer, Adrien, "Rôle of Advanced Maternal Age in Causing Mongolism of Children," *American Journal of Diseases of Children*, 1938, 55:79-92. See also Beal, G., and Stanton, R. G., "Reduction in the Number of Mongolian Defectives," *Canadian Journal of Public Health*, 1945, 36:33-37 (predicted on the age factor).

²⁴ Wallin, "Mongolism Among School Children," p. 110.

adequate proof. The following list includes the factors most frequently mentioned.

(1) Age of the mother.

(2) Maternal alcoholism, tuberculosis, or syphilis, or the use of poisonous contraceptives. Some of the mongolism stigmata resemble those of congenital syphilis.

(3) Mental distress or emotional upsets during pregnancy.

(4) Defective heredity, suggested by the presence of psychopathic traits, or mongol stigmata or mongolism in some relatives. Mongolism has been genetically produced by the presence in the germ cell of five pairs of recessive factors, or two dominants and four pairs of recessive factors (Madge T. Macklin) or by the inheritance of two dominant genes (Penrose). Rosanoff, Handy, and Plesset believe germinal factors are the sole cause of mongolism.²⁵

(5) Mongolian traits spring from an infusion of Mongolian blood into the occident—mongolism is a racial reversion phenomenon—a fantastic theory advanced by Crookshank.²⁶ Mongolism occurs in all races, including American Indians, Negroes,²⁷ and Jews, and its blood type is similar to that of non-mongol defectives.

(6) The numerous biochemical studies that have been made seem to show little of major significance so far as clarifying the mode of causation of mongolism, except, perhaps, a low basal metabolic rate and low cerebral oxygen utilization.

(7) Increased pressure upon the fetus during the sixth and seventh weeks by an abnormally small amniotic membrane (fetal sac) is posited by William Van der Scheer and Jansen as a cause.

²⁵ Rosanoff, Aaron J., Handy, Leva M., and Plesset, Isabel R., "The Etiology of Mental Deficiency with Special Reference to Its Occurring in Twins," p. 130. See also Gates, Reginald Ruggles, *Human Genetics*. New York: The Macmillan Company, 1946, Vol. II, pp. 1111-1119 (references).

²⁶ Crookshank, Francis G., *The Mongol in Our Midst* (3d edition). London: Kegan Paul, Trench, Trubner & Co., 1931.

²⁷ Thompson, W. H., "A Study of the Frequency of Mongolism in Negro Children in the United States," *Journal of Psycho-Asthenics*, 1939, 1:91-94.

(8) Uterine exhaustion from advanced age, excessive child-bearing, or illness (Shuttleworth).

(9) Markus Engler asserts unqualifiedly "that the pathological condition of the uterine mucosa immediately preceding pregnancy is the only cause of mongolism." This condition can be induced by lead, iodine, phosphorus, quinine, syphilis toxin, tuberculosis, inflammatory conditions, the irritant action of a number of abortifacients, X-rays, and curettage (Ernest Mayerhofer's "curettage Mongols" and Röntgen-Mongols). The worst injury is produced by mechanical intervention from curettage (scraping the uterine lining).

(10) Ingalls posits "damage to embryonic tissue" occurring "between pre-embryonic and early embryonic life" (probably before the third month of the affected person), possibly due to some undesigned "maternal illness." He finds no evidence in favor of an endocrine basis.²⁸

(11) Degeneration of the corpus luteum of the ovary or disease or dysfunction of the ovary, suggested by the bleeding, prematurity, miscarriages, and the long interval before the birth of a mongol child. The period of sterility amounted to five or more years in 27.6 per cent of mongols as compared with 10.3 per cent for nondefectives in the Canadian study cited. Of the 255 mothers studied by Benda 64 per cent had no children after the birth of the mongol.

The functioning of the corpus luteum (the "yellow body" in the ovary which secretes the hormone) is dependent on the activity of the thyroid, pituitary, and adrenal glands. Change in the ovum before fertilization caused by a "temporary defect or dysfunction of the endocrine glands of the mother and by some nutritive deficiency" is the explanation offered by Tredgold.

(12) Thyroid disorder in the mother: 43.1 per cent of the

²⁸ Ingalls, Theodore H., "Pathogenesis of Mongolism," *American Journal of Diseases of Children*, March, 1947, 279-292; see also "Etiology of Monogolism: Children, August, 1947, 147-165.

mongols were born in areas of high incidence of thyroid disorders, as reported in the Canadian study. Myers deduces from the fact that 27 per cent of the mothers of mongols suffered from "acute nervous excitement" as compared with only 3 per cent of the control group, and from the fact that experimental hyperthyroidism in animals greatly reduces fertility the theory that mongolism is related to "some form of disturbed thyroid function in the mother."²⁹ Benda considers hypopituitarism of the mother as the most important factor.

(13) Defects of the thymus gland or the gonads, the thyroid (fetal hyperthyroidism), polyglandular dysfunction, or a hormonal deficiency of some undiscovered endocrine gland.

(14) Defective functioning of the pituitary gland in the fetus is the most frequently postulated glandular involvement. Benda, after years of investigation of the problem, states unequivocally that the mongol is a "pituitary cretin" produced by "congenital hypopituitarism." "All the facts gathered by us in the last years point to a pituitary disorder."³⁰ The hormone deficiency is traced to the anterior lobe of the pituitary, but does not involve the pure growth hormone. The hypopituitarism is related to deficiency in the basophiles (certain basic-staining cells of the anterior pituitary lobe). "Typical of the condition is the lack of basophiles."³¹ That the disorder is related to hypofunction of the pituitary gland is also the conclusion of Bixby, based on the glucose tolerance test and the low metabolic rates.³² Apparently the primary disturbance is centered in certain microscopic cells in the anterior pituitary, and the thyroid, and possibly other endocrines, are also often involved as secondary concomitants.

²⁹ Myers, C. Roger, "An Application of the Control Group Method to the Problem of the Etiology of Mongolism," *Journal of Psycho-Asthenics*, 1938, 142-150.

³⁰ Benda, C. E., "Further Clinical and Pathological Studies of Mongolism."

³¹ Benda, C. E., "Studies in the Endocrine Pathology of Mongoloid Deficiency," *Journal of Psycho-Asthenics*, 1938, 154. In *Mongolism and Cretinism*, pp. 129 f. the involvement of other cells is postulated (e.g., the eosinophils, which stain pink with eosin).

³² Bixby, Emily M., "Biochemical Studies in Mongolism," *Journal of Psycho-Asthenics*, 1938, 1:59-70

Chapter 12¹

GROWTH ANOMALIES RELATED TO ENDOCRINE DISTURBANCES

Fröhlich's syndrome or "pituitary infantilism"

Pronounced underfunctioning of the posterior lobe of the pituitary gland (hypopituitarism) before adolescence gives rise to an entirely different kind of disorder of development known as Fröhlich's syndrome (after Alfred Fröhlich, pharmacologist),² or dystrophia adiposogenitalis, or pituitary infantilism (an older designation). The pituitary gland, also called hypophysis, is a bilobular gland about the size of a hazel nut located at the base of the brain in a protecting saddle-shaped bony structure called *sella turcica*. This relatively rare disorder is characterized, first of all, by a slight diminution of the stature and of the hands and feet. The face is usually normal in appearance. A much more important characteristic is the excessive accumulation of fat, not over the limbs, but over the central portion of the body. The marked, so-called "girdle type" obesity occurs over the lower abdomen, hips, and breasts, giving a boy a feminine outline and a "sissy" appearance. The fat often hangs in big chunks and large folds, as shown in Fig. 9. This condition is referred to as pituitary obesity. The third characteristic, often pro-

¹ Appreciative acknowledgments are expressed to George A. Jervis, M.D., Director of Research, Letchworth Village, New York, for a critical review of Chapters 12 and 13 and for sundry suggestions and additions.

² A syndrome is a group of clinical symptoms characteristic of a disease.

nounced, is hypogenitalism³ or genital infantilism (lack of development of the genitalia and secondary sex characteristics, ordinarily accompanied by amenorrhea, frigidity, and sterility, and sometimes homosexual practices). The skin is usually soft, delicate, and hairless, but it may be dry. The hands are ordinarily pudgy and fingers tapering. The facial and body appearance is infantile or juvenile and feminine. The condition is accompanied by increased sugar tolerance and headaches when the gland is tumorous. The condition may be due to a constitutional defect or it may follow an infectious disease, such as encephalitis lethargica. The mental picture is one of sluggishness, phlegmatism, sleepiness, and psychomotor and mental retardation. These children readily fall asleep, even in the classroom. Charles Dickens's description of somnolent Joe applies to some torpid, corpulent children who may be cases of posterior lobe hypopituitarism: "Joe! Joe! Damn the boy, he is asleep again." Ordinarily calm, cheerful, and contented, but passive, submissive, and compliant, they may react to their structural defects—for ex-

³ Hypogenitalism or hypogonadism is a condition observed in the adolescent male and, less typically, in the female, apart from the Fröhlich syndrome. It is characterized by manifestations of incomplete sexual development. The patients are usually tall and thin, muscular development is poor, the genitalia are small, and the secondary sexual characteristics are lacking or undeveloped. Nervousness, various somatic complaints, shyness, and persistence of infantile emotional reactions are characteristic traits of the mental make-up. Intellectually, these persons are often below normal, although the lack of drive and emotional blocking are more responsible for poor scholastic achievement than actual intellectual deficit. Substitution treatment with sexual hormones results often in improvement of physical and mental manifestations. See Engelbach, William, *Endocrine Medicine*. Springfield, Ill.: Charles C. Thomas, 1932, Vol. III, p. 140; Le Marquand, Horace S., and Tozer, Frederick, H. W., *Endocrine Disorders in Childhood and Adolescence*. London: Hodder & Stoughton, 1943.

Various kinds of hypogonadism exist, depending upon the age of onset, severity, and seat of the disorder. Eunuchoidism (the condition of a eunuch) is caused by atrophy of the testes or castration (eunuchism) prior to, or at the onset of, puberty and is marked by a high-pitched, falsetto voice, scarcity of hair on the face and body, change of body proportions, and lack of gonadal secretion. See Nelson, Warren O., "The Physiological Basis of Hypogonadism," *The Medical Clinics of North America*, January, 1948, 32:97-121; Thompson, Willard O., "Endocrine Problems During Adolescence," *ibid.*, pp. 140-150 (discusses the effects of disorders of nearly all the endocrine glands).



FIG. 9. Pituitary adiposity (*adiposogenitalis*), Fröhlich syndrome. Age 17, height 60 inches, weight 228 pounds. The normal weight of a 17-year-old girl 60 inches tall is 109 pounds. Binet age eight, I.Q. 50.*

ample, the genital infantilism and feminine type of adiposity on the part of boys—by feelings of timidity, shyness, distrust, sullenness, and hostility. The feelings of resentment and frustration not infrequently engender efforts at compensation through exhibitions of aggressiveness or antisocial behavior.

Although some of these cases are mental defectives, usually of the higher levels (sporadic cases are found in the institutions and classes for mental defectives),⁴ and some are mentally and emotionally infantile (hence "pituitary infantilism"), many appear duller than they actually are because of their listless, sluggish responses. Levy classified only 5 of 33 Fröhlich boys (functional cases, diagnosed largely on the basis of fat distribution) at the New York Institute for Child Guidance as below average in intelligence, and 17 as above average.⁵ The Stanford-Binet I. Q.'s

ranged from 70 to 140. The median was from "110 to 119" as compared with a "median quotient of 99" for the entire

⁴ Whether the mental defect in these and other endocrine cases is primary or secondary to the endocrine defect has not been definitely determined. In some cases the mental defect is probably of combined primary and secondary genesis.

⁵ Levy, David M., "Aggressive-Submissive Behavior and the Fröhlich Syndrome," *Archives of Neurology and Psychiatry*, November, 1936, 991-1020.

* Photograph supplied by Walter E. Fernald, M.D.

group of behavior cases examined at the Institute. Twenty-six of the 33 were characterized as constitutionally submissive, 5 as aggressive, and 2 as mixed, whereas the majority of the other behavior cases were aggressive. Bronstein, Wexler, Brown, and Halpern,⁶ on the basis of a battery of psychological tests, classified 48 per cent of 35 obese children (24 boys and 11 girls) as superior, 25 per cent as average, and 25 per cent as below average. The I. Q.'s (based on the Stanford-Binet Form L) varied from 39 to 147, with a median of 109. Only 2 of 24 boys showed a tendency toward femininity. No endocrinological basis was found for their obesity. Molitch and Poliakoff,⁷ on the basis of many intelligence tests, classified three of their five Fröhlich cases as average, one as inferior, and one as subnormal. Incidentally, their anterior lobe hyper- and hypopituitary cases tended to be brighter (especially the hyper cases) than the nonglandular institutional inmates. Of the anterior lobe hyperpituitary boys, 18 per cent were classified as immature, 12 per cent as infantile, and 28 per cent as unstable. The corresponding percentages for the anterior lobe hypopituitary group were 8, 4, and 32. Although the pituitary cases were superior to the controls in the institution in school progress, they were inferior in general adjustment and behavior. Mental subnormality is not the rule among different kinds of pituitary cases.

Many investigators have concluded that the Fröhlich disorder is not caused solely by underfunctioning of the posterior pituitary lobe, but involves a lesion of the hypothalamus (a part of the brain which includes the pituitary gland), and that this lesion accounts for the obesity. Moreover, some authorities believe that too many children have been diagnosed as pituitary cases, especially Fröhlich cases. Thus no

⁶ Bronstein, I. Pat, Wexler, Samuel, Brown, Andrew W., and Halpern, Louis J., "Obesity in Childhood, Psychologic Studies," *American Journal of Diseases of Children*, February, 1942, 238-251 (references).

⁷ Molitch, Matthew, and Poliakoff, Sam, "Pituitary Disturbances in Behavior Problems," *American Journal of Orthopsychiatry*, January, 1936, 127 f.

endocrine disorders were found by Weiner in ten alleged Fröhlich cases,⁸ and by Bronstein, Wexler, Brown, and Halpern in 35 obese children. They attributed the obesity to sedentary habits, abnormal appetities, or other factors. Obviously the diagnosis of pituitary infantilism cannot be based solely on the presence of a mons-mammary-girdle type of adiposity; and functional types of pituitary disorders may not be revealed by present methods of diagnosis.

A number of writers (for example, Louis A. Lurie) report very favorable results from the treatment of young Fröhlich children with the appropriate hormones, such as pituitary and other glandular extracts. Others (for instance Hoskins)⁹ express scepticism regarding any favorable outcome of such treatment. The negative results from administration of pituitary extract to Fröhlich cases reported by Tredgold¹⁰ is corroborated by the author's experience so far as concerns mentally defective cases, the only kinds with which he has dealt.

Dwarfism

Types and causes. The generic term dwarfism is applied to persons of very diminutive stature irrespective of the causes of the deviation. Other terms applied to gross statural underdevelopment include nanism, nanosomia, microsomia, micromelia (short limbs), midgetism, and pygmyism. Persons who are markedly undersized fall into two main categories: those whose body proportions are diminutive but perfectly formed (these are referred to as midgets or ateleiotic dwarfs); and those whose body parts are disproportionate or asymmetrical. In addition, some dwarfs manifest various malformations or deformities of the head, face, spine—for example, hunchbacks, often victims of tuberculosis of the

⁸ Cameron, Alexander T., *Recent Advances in Endocrinology*. Philadelphia: The Blakiston Company, 1945, p. 352.

⁹ Hoskins, Roy G., *Endocrinology*. New York: W. W. Norton & Company, Inc., 1941, p. 181.

¹⁰ Tredgold, *Mental Deficiency*, p. 294. On the Fröhlich syndrome see Gates, *Human Genetics*, pp. 775 f.

spine—limbs—the bowed legs of the rickety type—and other body parts.

Some of the causes of extreme variations in stature are known; others are still a matter of conjecture. Types doubtless exist that have not yet been properly differentiated. Some types are produced by processes of stunting, possibly from deficiency in the growth hormone, lack of thyroid secretion in early life (as in cretinism), pituitary disturbances (as in the Lorain-Levi type, and possibly mongolism), other endocrine disorders, severe fetal or early life malnutrition, as in rickets (rachitis, which arrests the process of cartilage ossification when the tissue is about to ossify), and tuberculosis of the spine, congenital syphilis, congenital heart disease, and chronic kidney disease (nephritis).¹¹ Some types apparently represent extreme hereditary variations from the racial norm, possibly explicable as Mendelian recessives, dominants, double dominants, or mutations.¹² The pygmies represent normal racial or ethnic types and are not the result of pathological processes. The ancient Romans allegedly practiced artificial dwarfing.

Pygmies. Three types of pygmy tribes or races have been recognized, but some of the descriptions are rather discrepant.

(1) The African or Negrillos, found in various parts of Africa (especially in the equatorial forest regions), are said to be the smallest known human race, varying from 33 inches to four feet four inches (the Wambutti tribe). The skin color varies from reddish brown to yellowish brown; a heavy growth of hair covers the body and face; the forehead is retreating and the lower jaw protruding (prognathous); the fingers are long and tapering; and the feet are arched. A tribe of pygmies, one of the most primitive types of humans, culturally considered, was discovered in 1895 on the east side of the upper Nile.

¹¹ Glass, S. J., "Dwarfism Associated with Microcephalic Idiocy and Renal Rickets," *Journal of Clinical Endocrinology*, 1944, 4:47-53.

¹² For explanation of recessives, dominants, and mutations, see pages 206 ff.

(2) The Negritos (Spanish for little Negroes), found on Luzon and other islands of the Philippines, S.E. Asia, the Malay Archipelago, Polynesia, and other islands in the east Pacific, are weaklings of dwarfish stature. They have brown, black, or yellowish skin, flat noses, thick lips, short, broad heads, and closely curling hair. Some writers believe that they are of African origin.

(3) The wavy-haired Asiatic pygmies, found in the southern Malay Peninsula, in the Celebes, and in some East Indian jungles and other places, are taller than the African pygmies and have differently shaped heads (long or dolichocephalic). According to one hypothesis, these pygmies are survivors of a formerly widespread pre-Dravinian race. The Dravinians were the oldest of the East Indian races which dwelt in southern India.

Midgets. The normally proportioned midget type of dwarf is found sporadically in the United States, Europe, and other countries (see Fig. 13). Growth proceeds relatively slowly in these cases, the full height being reached later than is the case among normal persons. Many midgets throughout the centuries have been cynosures in the entourages of kings and queens; they have functioned as favorite pages, attendants, entertainers, or tutors in the courts of royalty or in the homes of the rich. Many have been exhibited as freaks of nature in museums, carnivals, sideshows, and circuses.

Attendants at the court of the Pharaohs included members of the Akka pygmies from equatorial Africa. Philetas of Cos (about 330 B.C.), tutor of Ptolemy Philadelphus and grammarian and poet, was so small that he is jocularly reported to have worn leaden shoes to prevent his being blown away by the wind. He may have been of the achondroplastic type. Julia, the niece of Augustus, had a male and a female dwarf, each two feet and four inches tall. Queen Mary I had a page who was two feet tall. Edward VI's "gracefully proportioned" dwarf, Jeffery Hudson (1619-1682), born of normal parents, was scarcely 18 inches at the age of nine, but eventu-

ally attained a height of three feet nine inches. When this noted dwarf was 22 inches at the age of twenty-five he was immediately adopted by Henrietta Maria of France as he "stepped out of a pie" at a dinner given by the Duke of Buckingham to Charles I. "Strenuous Jeffery," as he was known because of his energetic activity, eventually became a "captain of horse." He fought a duel with Crofts, whom he shot dead from horseback, so he would be on the level of his antagonist. After having been prisoner of the Dunkirkers of France and of Turkish pirates, he eventually returned to England, where he lived on a pension until his death. Among Henrietta Maria's other dwarfs were Richard Gibson (who died in 1690 at the age of seventy-five) and his wife Anne, whose combined height was seven feet two inches. Richard began as a page and eventually became miniature painter to Charles I and drawing master to the young daughters of James II, Queen Mary and Anne. This couple had nine children of whom the five who survived were of normal stature.

The most famous European dwarfs, the hunchbacks of Philip IV of Spain, were immortalized by the famous Spanish painter Velasquez.

Richebourg, the 23-inch Parisian dwarf, who died at ninety in 1858, was slipped through the lines as an infant in a nurse's arms during the Revolution and carried secret messages in his infant wrappings in and out of Paris. The handsome Polish dwarf, Borulwaski (1739-1837), who was a wit and "something of a scholar," stood 17 inches at six and 39 inches at thirty. The Austrian "dwarf-giantess," Mary Youngman, measured 35 inches at fifteen, was three feet and six inches round the shoulders, four feet three inches round the waist, and two feet about the legs.

The best known American midget was "General Tom Thumb" (Charles Sherwood Stratton), who was born in Bridgeport, Connecticut, in 1838 and is buried there (died 1883). He was exhibited for years throughout the United States and Europe by Phineas T. Barnum, the showman.

When first presented, he was less than two feet tall and weighed only 16 pounds. At twenty-five he had reached a height of 31 inches. In 1863 he was married to the diminutive Lavinia Warner. (See Fig. 10.)

The shortest adult dwarf on record apparently is the female Hilany Agyba, of Sinai, who was only 15 inches, which is about three inches less than the length of the normal infant at birth.¹³

The available data do not justify any positive conclusion regarding the causation of the growth deviation in these and some other cases of midgetism. They may represent different morphological types.¹⁴ Nor can definite conclusions be formulated regarding the question of how the distributions of intelligence and other mental characteristics among midgets differ from the corresponding distributions in the general population, because of the paucity of the experimental data. Although many midgets are bright, clever, nimble, witty, and resourceful (in contrast with many giants), and perhaps also sensitive and vengeful, some are dull, apathetic, or mentally deficient and require special educational adjustments. The undesirable personality reaction patterns sometimes induced by a morbid consciousness of diminished stature may also create special problems of adjustment. Although they are rarely encountered in the public schools, the writer has observed a number of midgets and dwarfs of various kinds in the public schools in the middle west and in the east, only a few of whom were referred for special class assignments.

A less well recognized type, the "Brissaud type" of pituitary dysfunction, deserves a passing reference because of its association with mental defectiveness. In this small group manifestations of thyroid deficiency are superimposed upon the characteristic hypopituitary picture. Stature is below

¹³ Consult the tabular data reproduced in Teagarden, Florence M., *Child Psychology for Professional Workers*. New York: Prentice-Hall, Inc., 1946, p. 134.

¹⁴ See Dupertuis, C. Wesley, "The Size and Proportions of Adult Midgets," *American Journal of Physical Anthropology*, N. S., 1945, III, 111-127.

normal, genital organs underdeveloped, and the face somewhat cretinoid in appearance. These patients are usually mentally defective, in contrast with the pituitary individuals. Some benefit may be obtained from early and continuous thyroid treatment.

Achondroplastic dwarfs. The muscular, strong-man type



FIG. 10. General and Mrs. Tom Thumb, noted American midgets of Barnum Circus fame.*

* From Major, Ralph H., *Physical Diagnosis* (2d edition). Philadelphia: W. B. Saunders Company, 1940.

of dwarf is well known from representations in Egyptian architecture and in legend (the Scandinavian gnomes) and from circus and carnival exhibitions. The Egyptian deities Bes and Ptah and Chnoum-hotep (about three feet tall at twenty, Fig. 11), Attila, King of the Huns, and Wladislaus ("Cubitalis"), King of Poland, have been classified as achondroplastic dwarfs, a name first applied to the group by Parrot in 1878. The condition had previously, in 1860, been mistakenly diagnosed by Müller as "fetal rickets," although the trunk and limbs in rickets are symmetrically proportioned. Many other terms for the condition are in use, including Ollier's disease and micromelia (*melos*, limb).



FIG. 11. The Egyptian dwarf Chnoum-hotep, about 3 feet tall at the age of 20.*

The majority of these dwarfs die in utero at about the eighth month. Some of those who survive die in early childhood, but others continue vigorous and normally healthy until old age.

The most distinctive characteristic of achondroplasia, which does not show any racial or sex predilection, is the lack of normal symmetry between the length of the limbs and the torso (trunk), as seen in Figs. 11 to 13. The vertical midpoint of the normal body falls at about the pubic bone. In the achondroplastic the proportions above and below this point are about 2 to 1, because of the short limbs. The total stature varies from about 36 to 56 inches. Mörch's means are 47.2 inches for women and 51.2 for men. Al-

* Photograph from Ralph H. Major, M.D.; reproduced from Ruffer, Marc Armand, *Studies in the Paleontology of Egypt*. Chicago: University of Chicago Press, 1921.

though large differences exist among different achondroplastics, in general the trunk is about normal in length (see Fig. 12), while the arms and legs are very short, thickened, hard, and muscular, with deformities in the shaft. Enlargement at the joints, knock knees, and bow legs are frequent, and produce a waddling gait. Ordinarily, the hands are broad and short and the fingers, all of about the same length, spread out radially (trident-shaped, Fig. 14). The spine may display a lateral or anteroposterior curvature (a lumbar lordosis or kyphosis) and the abdomen may protrude. The skull appears large and high (macrocephalic and brachycephalic), and shows enlargement of the frontal and parietal bosses, premature union of the bones at the base, and a small sella turcica (the depression in the sphenoid bone that contains the pituitary gland and circular sinus). The hair on the scalp is heavy and silky. The forehead is high and broad and the nose pug-shaped, with a depressed bridge and large nostrils. The condition is frequently attended by a mild hydrocephalus of the stationary kind and by precocious and excessive sexuality.

Although the cause of the condition is not definitely known, it starts in early intrauterine life. Some growth process interferes with the development of

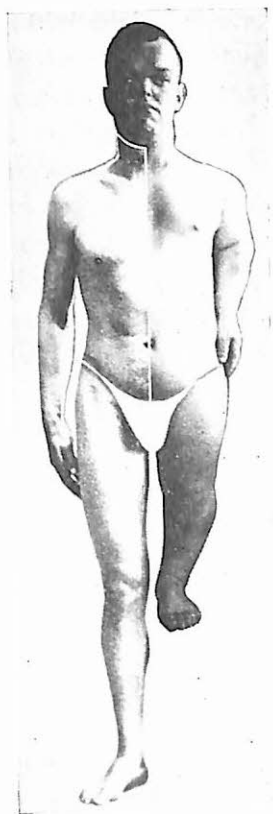


FIG. 12. A photographic chimera. The normal body proportions on the left are contrasted with those of the achondroplastic dwarf on the right. Note the congruence of the trunk and head and the shortness of the limbs in the achondroplastic.*

* Courtesy of Mörch, Ernest T., "Inheritance of Achondroplasia," *Journal of Heredity*, October, 1940, p. 443.

the cartilage part (*chondros*, cartilage) at the end of the long bones and causes premature union of the epiphyses (bones that are separated from the long bones in early life). The growth interference and distortion of these osseous processes have been ascribed to the smallness of the sac (amnion) that encloses the fetus (J. Jansen), to fetal rickets (Müller), and to the abnormal functioning of one or more of the endocrine glands. None of these and other hypotheses have been confirmed. A number of pedigree studies have suggested that the condition may be inherited as a single or double Mendelian dominant, a Mendelian recessive, an independent mutation or sport, or a chromosomal variation. Mörch maintains that many cases have been misdiagnosed and concludes from the examination of bona fide cases that the condition cannot be explained as a Mendelian recessive but rather as a dominant, and that many cases are mutations. Thirty-nine of his cases were classified as "isolation" types, as against only four familial types, occurring in two consecutive generations.¹⁵

The condition was reported for the first time in June, 1939, in purebred rabbits of Havana stock. An extensive investigation of 228 achondroplastic dwarf rabbits, all of whom were born dead or died a few hours after birth (they are homozygous¹⁶ for the factor and do not survive) showed that the outstanding characteristics in rabbits are about the same as in achondroplastics in human beings and in other animals, namely: greatly reduced size (the mean weight being 63.2 per cent of that for the normal young); markedly shortened limbs; large abdomen; squarish head and broad, short face; deficient ossification and cartilage formation; and a transverse depression of the root of the nose. The abnormality does not involve the genitalia and it is not sex-limited, although it is more prevalent among females. The transmis-

¹⁵ Mörch, Ernest T., "Achondroplasia Is Always Hereditary and Is Inherited Dominantly," *Journal of Heredity*, October, 1940, 439-444 (references).

¹⁶ Homozygous organisms have a pair of identical genes from the eggs and the sperm for any given character. Like contributions have been received from both parents.

sion was found to follow the simple recessive ratio of 25 per cent. Vitamin deficiency may be a factor in the causation, but the defects in the thyroid (colloid deficiency) and pituitary (increased basophiles and acidophiles) are interpreted as results, not causes, of the condition.¹⁷

The parents of achondroplastic children are often mentally and physically normal. Achondroplastics are usually normal in sex development, are fertile, and may produce both normal and dwarfed children.¹⁸ Many midgets are also fertile. Two midgets have been known to be parents of midgets, just as two achondroplastics have had achondroplastic children. The births usually require caesarian deliveries because of the smallness of the pelvis.

During the first year or two the distinction between achondroplasia, rickets, and cretinism often cannot be made without the aid of X-rays of the bones. The epiphyses consolidate very early in achondroplasia, but very late in cretinism. Moreover, the untreated cretin is ordinarily slow, lethargic, stupid looking, and mentally defective, whereas the achondroplastic dwarf tends to be bright looking, active, and energetic. In time the unsymmetrical dwarfism of achondroplasia is readily distinguished from the symmetrical underdevelopment of the rachitic and cretinoid dwarfs. The statural differences between a cretin, a midget, two achondroplastic dwarfs, and a normal male adult are shown in Fig. 13.

Among the distinguishing characteristics of the rickety dwarfs are chicken breast (in which the breast bone is prominent), a high (olympic) forehead, a natiform (buttock-like) skull, marked deformities of the legs, scoliosis (lateral curvature of the spine), and the rickety rosary (a row of projections like beads at the junction of the ribs and the cartilages). A

¹⁷ Brown, Wade H., and Pearce, Louise. "Hereditary Achondroplasia in the Rabbit," *Journal of Experimental Medicine*: "I, Physical Appearance and General Features," 1915, 241-260; "II, Pathologic Aspects," 1915, 261-280; "III, Genetic Aspects: General Considerations," 1915, 281-295.

¹⁸ Speck, George. "Pregnancy in Case of Pituitary Dwarfism," *American Journal of Obstetrics and Gynecology*, February, 1916, 51:217-220. "True dwarfs often have normal babies."

rare form of dwarfism of unknown origin, known as progeria, is characterized by pronounced underdevelopment of the stature and genitalia and premature senility. These children

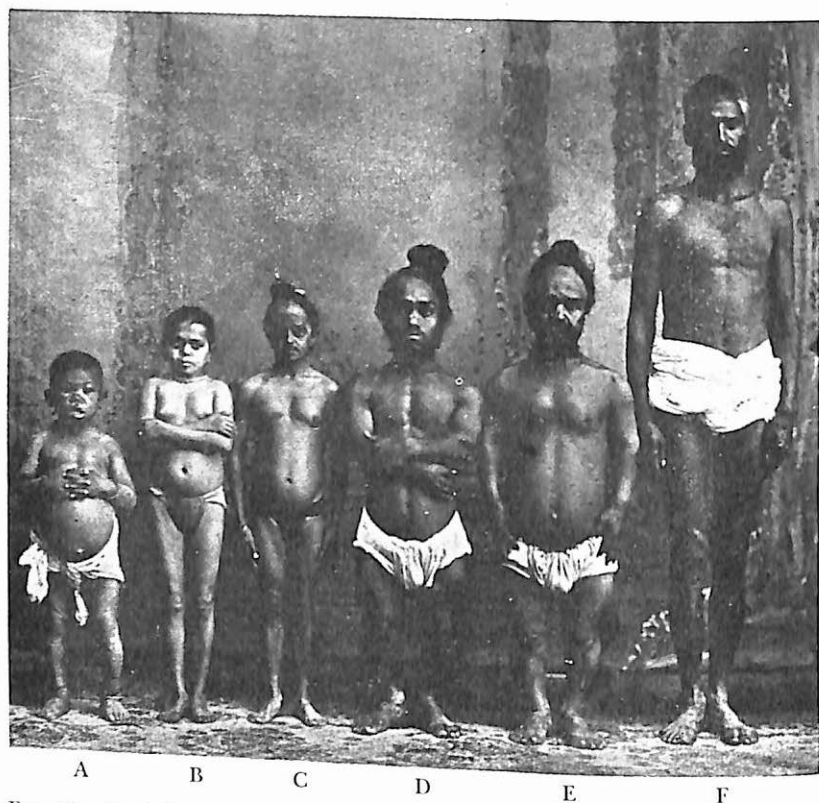


FIG. 13. Varieties of dwarfs among East Indians: (A) cretin; (B) and (C) midgets (the condition is also referred to as Gilford's ateleiosis; it bears resemblance to Lorain-Levi dwarfism); (D) and (E) achondroplastics; (F) normal adult.*

appear like little men or women in general and facial appearance. More psychometric studies are needed to determine the distribution of the intelligence level among these children who became prematurely old.

* From Rischbiet, H., and Barrington, Amy; reproduced in Major, Ralph H., *Physical Diagnosis* (3d edition). Philadelphia: W. B. Saunders Company, 1945, p. 44.

No treatment exists for achondroplasia, other than overcoming localized limb deformities, knock knees, and bow legs by proper orthopedic procedures and, possibly, the treatment of progressive hydrocephaly by surgical intervention.

The proportion of mental deficient and retardates among achondroplastic dwarfs has not been satisfactorily determined. There is a dearth of psychological studies.¹⁹ Many have made normal progress in the regular curriculum. Such pupils should be treated as normal persons and not as curios. No attention should be directed to their physical anomalies and they should not be subjected to invidious comparisons or derisive comments. Many have rated as normal or above normal in energy, enterprise, and foresight in their occupational and business pursuits. Most of the circus specimens seem to be alert and to react in a fairly normal manner. Nevertheless, some are mentally limited in varying degrees and require special educational adjustments. Those who gravitate into the institutions for mental defectives usually rate among the higher grade cases, such as J. F. in Fig. 14.

ILLUSTRATIVE CASE of achondroplasia. The achondroplastic dwarf shown in Fig. 14 (J. F., right side), taken at the age of 23-3, was admitted to the residential school at 17-6, with a standing height of 41½ feet, weight 102½ pounds, small square hands, short stubby fingers, short feet, all toes about the same length, a large head (circumference about 24 inches), and well developed muscles. His Form L Binet at the age of 17-8 was 10-4, I. Q. 69; at age 20-8, 10-2, I. Q. 68; and at 22-9, 12-0, I. Q. 80. At the age of 18-6 he had reached the seventh grade, but was doing only about fifth-grade work. The following year he was started in the band, where he continues as the bass drummer. He is the "brightest boy in the print shop" where he is a compositor and serves as boss. He is an "accomplished worker," even-tempered, affectionate, and cheerful. There is "no child here with a better disposition; his sunny disposition is a legend at the institution."

This achondroplastic dwarf is contrasted in Fig. 14 with a

¹⁹ See, however, Foster, Roberta, Brown, Andrew W., and Bronstein, I. Pat. "The Mental Development of a Group of Dwarfish Children," *Journal of Psycho-Asthenics*, 1939, 2:143-153.



FIG. 14. W. L. (left), a dwarf with Pott's disease, possibly with pituitary involvement, and J. F. (right), an achondroplastic dwarf.*

* Courtesy of E. Arthur Whitney, M.D., Superintendent, Elwyn Training School.

FIG. 15. Two types of pituitary growth disorders contrasted. On the left, R. S., a Lorain-Levi dwarf (undersecretion of anterior lobe of pituitary in early life), at age 6-7, weight 29 pounds, height 33.7 inches, Stanford-Binet I. Q. 79. On the right, M. K., Fröhlich syndrome or adiposogenitalis (underfunctioning of posterior pituitary lobe before adolescence), at age 6-2, weight 78.5 pounds, height 49 inches, Stanford-Binet I. Q. 79. At the age of 11-6 her weight was 141, height 55.5 inches, Stanford-Binet I. Q. 71.†

† Courtesy of Louis A. Lurie, M.D., Director, Child Guidance Home, Cincinnati.



dwarf, W. L., who became a victim of Pott's disease (tuberculosis of the spine) when young and entered the same institution at the age of 9-1. His photograph was taken at the age of 34-9. His head circumference at the age of 24-8 was only 20½ inches. His head is round and hands and feet are small. At the age of 19-10 his Stanford-Binet age was 7-6 and I. Q. 47; at 26-0, the Form L Binet age was 7-4, I. Q. 49. He was reported to have reached his limit in school at the age of twenty, reading at the fourth-grade level. For several years he was reported to be deteriorating, with growing deafness. He has for many years been very helpful as the errand boy in the infirmary—the "grandfather of the infirmary," who is very popular and "everybody's friend." He is withal a "miser" who saves his pennies.²⁰

Lorain-Levi dwarfs

The Lorain-Levi dwarfs (see Fig. 15) represent a symmetrical type of dwarfishness produced by serious undersecretion in early life of the growth hormone (somatropin) of the anterior lobe of the pituitary gland (hypopituitarism). Mature midgets of this kind are miniature adults of normal body proportions who present a graceful appearance. All body parts are small, including the face, head, hands, and feet. The fingers are narrow and tapering, and the hair soft and silky. If the sex (or gonadal) hormone is also involved, the genitalia are infantile and the secondary sex characteristics remain largely underdeveloped. Sterility in women and impotence in men are common sequelae. The term pituitary infantilism has been applied to those undersized from birth, but this term might better be reserved for early cases of posterior lobe hypopituitarism. Some pituitary dwarfs, however, tend to retain childish personality traits and constitute cases of psychic infantilism. Their immature facial expression (facial juvenilism) assumes a wizened appearance as they undergo premature aging. Adequate data are not available on the distribution of intelligence based on intelligence

²⁰ Unfortunately, it has not been possible to obtain the statural measurements of this case nor the sitting and standing heights and arm lengths of these two dwarfs and the pituitary giant in Fig. 16.

tests, among these midgets, but mental deficiency is not a marked feature of the syndrome. Although some may be mentally retarded, the majority are alert, intellectually normal, or bright. They tend to be somewhat aggressive, probably as a protective defense against their diminutive stature.

The undersecretion of the growth hormone may be due to congenital hypoplasia (incomplete development), or to an early lesion of the gland produced by an infection or by a tumorous growth. Injection of the missing growth hormone (possibly in combination with the sex or gonadotropic factor) has not proved very efficacious; it may increase the height from only six to nine inches. Although many of these midgets die young from some intercurrent disease, others live on into old age.

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Pituitary or acromegalic gigantism

Characteristics and causation. The growth disorder at the opposite pole of achondroplastic dwarfism is pituitary or acromegalic gigantism (*akron*, extremity; *megas*, large). Excessive secretion of the growth hormone (or of the acid-staining cells known as acidophiles) of the anterior lobe of the pituitary (hyperpituitarism) in childhood or adolescence before the calcification of the epiphyses results in this form of symmetrical overdevelopment. The height of the pituitary giant varies ordinarily from six and a half to eight feet. The limbs, hands, and feet are excessively long, with arm span exceeding the total stature and the lower limbs exceeding the trunk measurement. The thyroid is enlarged in about 50 per cent of the cases. The early stages are characterized by rapid growth, excessive muscular strength, and hypersexuality.

The terminal hypofunctional stage, after the disorder has burned itself out, is marked by muscular weakness, obesity, somnolence, genital regression, loss of sex power (gonadal atrophy) or amenorrhea, and sterility. Infantilism is a characteristic of some of the cases. Acromegalic gigantism should be distinguished from adult acromegaly (or Marie's disease, after Pierre Marie, French physician), a disease characterized by progressive enlargement of the face and head, hands and feet, and thorax. Acromegaly is gigantism of the adult and gigantism is acromegaly of the adolescent (Edouard Brissaud and Henri Meig, French physicians); that is, the result of overactivity of the anterior lobe in adolescence is gigantism, and in adulthood, acromegaly.²¹ A group of giants

²¹ See *Time* magazine for October 11, 1948, p. 92, for photographs of a man who was of normal appearance at 24. At 29, when acromegaly had set in, a broadening of the nose is apparent; three years later, at 32, the facial features had become coarse and heavy. Photographs of a dwarf 36 inches tall and a giant 9 feet 3½ inches tall are also shown.

(called "infantile" by Launois and Roy, French physicians) who possess very long legs, marked atrophy of the genitalia, and failure of the epiphyses to unite has been distinguished. The two types often merge. Doubtless other types of gigantism exist.

ILLUSTRATIVE CASES. The contrast between the dwarf with spinal tuberculosis and a pituitary giant is shown in Fig. 16. E. P., aged twenty-five at the time the photograph was taken, entered the Training School at the age of 15-6, when he was very tall, with conspicuously large hands, feet, and head (circumfer-

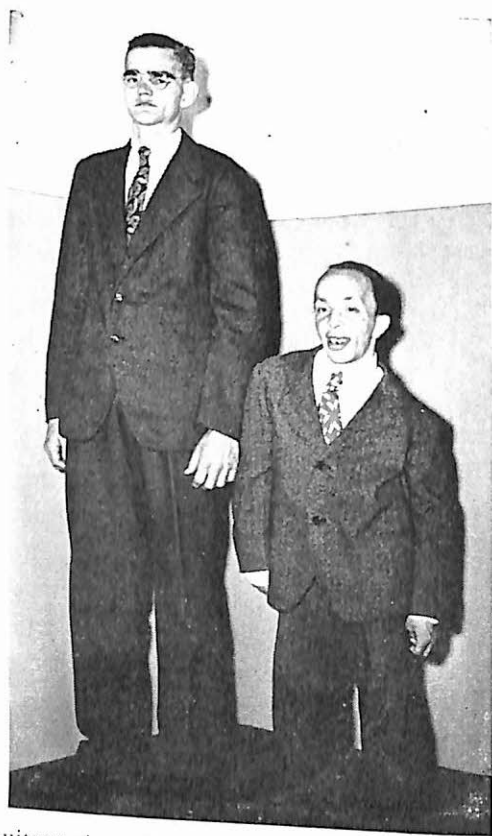


FIG. 16. A pituitary giant, E. P., contrasted with a dwarf with tuberculosis of the spine.*

* Courtesy of E. Arthur Whitney.

ence 23 inches). His Stanford-Binet age at the age of 15-7 was 7-4, I. Q. 47; at 23-9 his Form L Binet age was 7-2, I. Q. 48. A year after admission he was doing second grade work in reading, spelling, and arithmetic, was "very lazy," unambitious, resistant to instruction, "but was polite, friendly, and obedient." He has worked at making beds, dry scrubbing, and outside jobs, but is not of much account. He must be watched because he indulges in homosexual practices with small boys and shows undue interest in girls. Difficult birth required artificial respiration. The excessive growth may have been arrested by X-ray irradiation of the pituitary gland.

The largest giants in recorded history, without reference to type, include: the Chinese Chang, 8 feet; a skeleton in the museum at Bonn, Prussia, 8 feet; the skeleton of the Irish Charles Byrne (or O'Brien, 1761-1783) in the museum of the College of Surgeons of England, 8 feet 2 inches; the skeleton in the Museum of Trinity College, Dublin, 8 feet 6 inches; the Austrian Joseph Winkelmaier (1865-1887), 8 feet 9 inches; the famous Biblical giant, Goliath, whom David slew with a sling and stone, about 8 feet 9 inches, according to Josephus and the Septuagint, or about 9 feet 6 inches, according to I Sam. 17:4; the Russian giant Machnow, 9 feet 3 inches (see Fig. 17); and Topinard's Finlander, reputedly the tallest authentic case of gigantism in medical literature, 9 feet 4 inches. According to a press report on February 15, 1948, another Finn, Vaino Myllyrinne, was the "world's tallest man" at the time. He stood "9 feet and 1 inch with his shoes off and weighed 391 pounds" at the age of thirty-eight. In this country one of the tallest giants on record was Robert P. Wadlaw, of Alton, Illinois, who died on July 15, 1940, at the age of twenty-two from a foot infection caused by the chafing of an ankle brace he wore to support his weight. His height, according to one press report, was 8 feet 10.3 inches and his weight was 431 pounds on the preceding June 27, at a St. Louis hospital. According to another report his height was 8 feet 9 inches. He wore size 39 shoes. At birth his weight was nine pounds and at six months 30 pounds. His abnormal

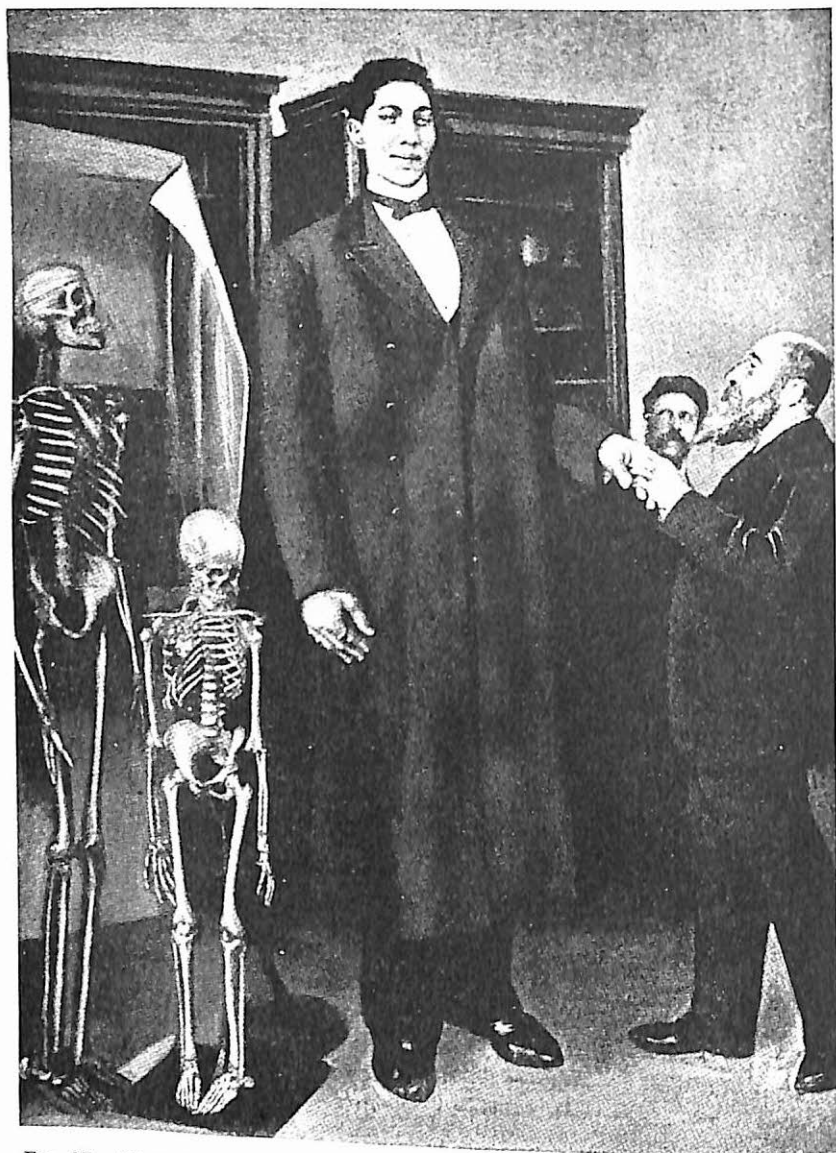


FIG. 17. The Russian giant Machnow, 9 feet 3 inches tall, contrasted with a man of normal stature, a tall Bushman (skeleton), and a Patagonian dwarf (skeleton).*

* Photograph from Ralph Major; reproduced from Launois and Roy, *Etudes biologiques sur les géants*. Paris: Masson et Cie, 1904.

growth acceleration was not noticed until he was one year old.

More experimental data are needed before broad generalizations can be formulated regarding the distribution of intelligence among pituitary giants. Some are normal, others are subnormal. E. P. clearly rates as a mental defective.

The treatment of gigantism, often unsuccessful, includes the administration of thyroid extract, sex hormones, X-ray irradiation of the pituitary gland, or the surgical removal of a tumor. Of course, gigantism exists as a racial trait, just as does pygmyism. Among giant tribes in Africa is the Watusi tribe which dwells at the northern end of Lake Kivu.

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Laurence-Moon-Biedl syndrome

This rare, imperfectly understood, heredofamilial condition (about 200 cases had been reported to 1940), which is apparent at birth and affects the sexes in about equal degree, was first identified by J. Z. Laurence and R. C. Moon in 1866, and was more completely described by Arthur Biedl in 1922. It is now known that many cases exist, sometimes referred to as "incomplete," that do not exhibit the five cardinal symptoms of the syndrome, namely, pigmentary degeneration of the retina (retinitis pigmentosa), leading to various degrees of blindness or night blindness, obesity, hypogenitalism (or genital dystrophy, or adiposogenitalism—arrested develop-

ment of the primary and secondary sex characteristics), polydactylism (extra digits) or syndactylism (united or adherent digits), and mental deficiency or retardation. Thus only 24 of the 102 cases culled from writings by J. Warkany, G. S. Frauenbuerger, and A. G. Mitchell manifested all of these symptoms. The remaining 78 showed wide variations, such as deafness, squint eyes, rapid, automatic movements of the eyeballs (nystagmus), atrophy of the optic nerve without the retinal pigmentation, congenital heart disease, and dwarfism. The most prevalent symptoms were, in descending order, obesity, retinal pigmentation, poly- or syndactylism, mental retardation, and genital dystrophy. Fifty-three showed an atypical Fröhlich syndrome (obesity and genital dystrophy), and only 11 a typical Fröhlich syndrome (obesity, dwarfism, and genital dystrophy). The thirteen-year-old boy and the twenty-year-old girl described by Lurie and Levy presented a syndrome of deafness without visual defect, genital dystrophy, dwarfism, increased sugar tolerance (the Fröhlich syndrome), mental retardation, syndactylism, and familial occurrence. The I. Q.'s for the boy and girl were 79 and 73 (Form M Stanford-Binet) and the S. Q.'s (social quotients, from the Vineland Social Maturity Scale) 98 and 80.²²

The exact cause of the malady has not been determined. Some have ascribed the obesity to malfunctioning of the pituitary or the thalamus, but this would not explain the associated congenital deformities. The variety and divergence of the symptoms have suggested the concurrent operation of a number of genetic factors (for example, Biedl, and R. L. Jenkins and H. G. Poncher),²³ such as one factor for the obesity, genital dystrophy, and dwarfism; a second factor for the retinal pigmentation; a third for the hand and foot deformities; and possibly a fourth, a cerebral-linked factor, for the mental retardation. The mode of transmission is believed to follow

²² Lurie, Louis A., and Levy, Sol, "Laurence-Moon-Biedl Syndrome," *Journal of Pediatrics*, December, 1942, 793-802.

²³ *Ibid.*

the pattern of a Mendelian recessive, but the hereditary antecedents may differ in different members of the fraternity.

No cure exists for this abnormality. Endocrine therapy does not improve the mental condition or remove any of the physical abnormalities, although the persistent use of thyroid extract may be of some value.²⁴ These children attend school, where they are rarely understood and often misunderstood, and frequently are unable to meet the scholastic requirements and fail of promotion because of mental deficiency, or retardation and sluggishness, or lack of drive. Many require the advantages afforded by the well conducted special class with its individualized, developmental activity program.

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Hypoparathyroidism and hyperparathyroidism

Severe underaction of the parathyroids, resulting in deficient calcium and phosphorus metabolism, produces nutritional disturbances, rickets, nervous irritability, muscle spasms, tetany, and sometimes convulsions with occasional fatal results. Partial degrees of parathyroid deficiency may

²⁴ Cameron, A. T., *Recent Advances in Endocrinology*, p. 355.

exist for months without detection and may be the cause of the child's restlessness, touchiness, emotional instability, and hypersensitiveness. The calcium-starved individual tends to become excitable, irritable, and cross. According to Lurie, conduct disorders in children are connected with calcium deficiency. Striking improvement in disposition and conduct has been recorded from the administration under medical supervision of parathyroid extract and calcium, single or in combination, and vitamin D, with sunshine and high calcium foods (milk and cheese), with the avoidance of phosphorus-containing foods and drugs (phosphorus depresses calcium and *vice versa*). Some types of mental defectiveness have been attributed to calcium deficiency; this conclusion requires experimental confirmation. Overaction of the glands may produce disturbances of the bony structures because of excessive withdrawal of lime salts from the bones, muscular weakness, and apathy, but without mental impairment. This condition is known as Recklinghausen's disease (osteitis fibrosa).²⁵

Thymus disorders

The functions of the thymus, a small bilobular gland, consisting mostly of lymphoid cells, located medially in the lower neck beneath the upper part of the sternum, have not been very definitely determined. The gland is active in early life and ordinarily atrophies or becomes functionless at about the age of thirteen. From this circumstance and other facts the conclusion has been reached that the gland serves as a brake on the development of the sex glands until puberty. It is generally thought to be related to the production of white corpuscles. "Thymic idiocy" in puppies has, it is alleged, supervened two or three months after its removal in the second week. Desire Magloire Bourneville believed that the

²⁵ Parathyroid disorders: Keating, F. Raymond, "Diagnosis and Treatment of Hyperparathyroidism," *Journal of Michigan State Medical Society*, October, 1946, 45:1349-1353.

absence of the gland could cause feeble-mindedness. He found the gland absent in 28 autopsies of mentally deficient children. In 408 autopsies at the Bicêtre of young "noncretinous idiots" it was present in only 104 cases. On the other hand, the thymus was demonstrated in the post mortems of all of 61 mentally normal children from one month to thirteen years of age in an investigation made elsewhere (Tredgold). According to more recent reports, a five-year-old mentally retarded child unable to speak was talking after daily injections for two months of the thymus extract developed by Adolph M. Hanson. Another child of less than a year grew six inches in six weeks from the same treatment. In spite of these positive indications, the experimental findings, which cannot be summarized here, are discrepant and it behooves the cautious student to maintain a sceptical attitude regarding the causal relationship of thymus defects to mental defects. The relationship may be one of concomitance rather than of causation. The thymus defect may be secondary to some more general aplasia in the mentally defective which is the main cause.

Precocious puberty (pubertas praecox)

Precocious abnormal sexual development. Many studies of pathological cases of precocious sex development (pubertas praecox) have conclusively shown that it is possible for secondary sex characteristics to mature and for the gonads to begin to function during the first few years of life in either sex. These cases of sex precocity are caused by overfunctioning in early life of some of the endocrine glands, due to tumors (neoplasms) or hyperplasia (abnormal multiplication of normal elements in an organ), especially of the gonadal gland (hyper-testicularism and hyperovarianism), the pineal gland, the adrenal cortex, and possibly the pituitary gland (excess secretion in early life of the master sex hormone) and the thymus. Some cases probably stem from pathological brain conditions, such as cysts in the ventricles, hydrocephaly, tuberous sclero-

sis,²⁶ and injury to the hypothalamus which may impair the inhibitory function of the pituitary gland.²⁷ Emil Novac maintains that the most common form of sex precocity is the constitutional type, in which a healthy girl without evidence of endocrine or other defects may bear children when she is between five and nine years of age, but does not differ from other girls after the age of ten or eleven. Such allegedly normal cases of extreme sex precocity, perhaps more common than usually assumed, are consistent with Sigmund Freud's doctrine of infantile sexuality, which, apparently, has been confirmed by an extensive investigation along nonpsychoanalytic lines of the sex life of 12,000 American males in all walks of life.²⁸ This investigation has also confirmed many other facts of sexual behavior well known from earlier less extensive investigations. By way of contrast, lack of secretion of the sex hormones in early life, or of hormones that stimulate the development of these hormones, retards or prevents sex development and may produce hypogenitalism, hypogonadism, pituitary eunuchism or eunuchoidism, as already pointed out.

The picture presented by excessive early hyperovarianism includes: (1) the beginning of menstruation during the first few weeks or months of life; (2) the development of the adult female secondary sex characteristics of face, form, and hair distribution, and (3) diminished stature. Signs of early hyper-testicularism may include: (1) precocious enlargement of the prostate and seminal vesicles and seminal discharges; (2) early male distribution of hair; (3) a deep voice; and (4) marked physical strength.

Early hyperactivity of the adrenal cortex in girls may re-

²⁶ Pratt, Jean P., and Schaefer, Robert L., "Precocity, Virilism, Adrenal Cortical Tumor," *American Journal of Obstetrics and Gynecology*, May, 1945, 49:623-633 (references; see remarks by J. G. Greenhill).

²⁷ Scholder, Bernard M., "The Syndrome of Precocious Puberty, Fibrocystic Bone Disease and Pigmentation of the Skin: Eleven Years' Observation of a Case," *Annals of Internal Medicine*, January, 1945, 105-118 (references).

²⁸ Kinsey, Alfred C., Pomeroy, Wardell B., and Martin, Clyde E., *Sexual Behavior in the Human Male*. Philadelphia: W. B. Saunders Company, 1948.

sult in the growth of a beard, masculine hair distribution, a deepening of the voice, marked muscular development and strength, a skeleton of adolescent proportions, overdevelopment of the clitoris, and sex precocity. Incidentally, it may be remarked that when the disease starts in later girlhood, the female primary and secondary sex characteristics fail to develop, except for hypertrophy of the clitoris. The characteristics of adult female virilism include excessive hairiness, muscular hypertrophy, regression of the sex organs, loss of the feminine configuration, hypertrophied clitoris, amenorrhea, and eventual sterility (adrenal virilism or adrenogenitalism). Virilism may also follow disorders of the pituitary and pineal glands and possibly of the thymus. Masculinism or virilism can be imposed on the feminine structure at any time. The antithesis of virilism—adrenal feminization—may occur in the male sex from excess of cortin. It is characterized by the development of the breasts, atrophy of the external genitals, loss of libido (sex drive) and potency, loss of hair, gain in weight, and the assumption of the feminine form.

ILLUSTRATIVE CASES of puberty praecox will lend needed concreteness. A five-year-old boy with a tumor of the left testis had grown rapidly, possessed a deep bass voice, and had developed a beard and pubic hair. At the age of nine he was 56 inches tall and weighed 97 pounds. Four months after the removal of the growth his beard disappeared, the genitals diminished in size, his emissions ceased, and his voice and mental characteristics became childlike again. (Joshua H. Leiner.) A girl with an ovarian tumor began to menstruate, developed breasts, and grew pubic hair at seven. A boy with a pineal disorder was subject to emissions and was markedly precocious mentally at 17 months; at 44 months he answered questions in a loud, bass voice, was independent and self-possessed with strangers, and spurned toys for small children (Leiner). Another boy of eight with a pineal disorder possessed the sex development and functions of a boy of fifteen or sixteen. An adrenal case, a mentally retarded, bad-tempered boy, was normal at six months. At twelve months he had a deep voice, large hands, great muscular strength, a dental age of about three years, a bone age of five years, and a sex de-

velopment of eight. His prostate was the size of a walnut and the penis was markedly enlarged. He practiced self-abuse frequently. He failed to survive the removal of a tumor the size of a golf ball from the right kidney (Fraser's case, 1940).²⁹ A girl, aged 4-6, with a cortico-adrenal disorder, had a markedly enlarged clitoris and required shaving because of the growth of a beard (William Bulloch and James H. Sequeira). Another girl of seven looked like a young man with silky black beard (E. E. Glynn).

The cortico-adrenal cases are five times as prevalent among females as among males (Norma V. Scheidemann), whereas the cases that originate in pineal or pituitary disorders are more numerous in the male sex (J. P. Greenhill). Overactivity of the adrenal cortex before birth may result in pseudohermaphroditism (for example, the possession of the external male genitalia and female ovaries and uterus).

Many observations and the few available examinations by means of objective psychoclinical tests seem to show that the precocious sexual development is not paralleled by a corresponding mental precocity. On the other hand, marked tendency toward mental retardation and immaturity (infantilism) characterizes the group as a whole. On the basis of the references (largely based on subjective impressions) to the mental status of 62 of 190 cases reviewed by Stone and Doe-Kuhlmann,³⁰ 21.3 per cent were classified as above average, 37.7 per cent as average, and 41.0 per cent as below average. Roger Williams, on the basis of the review of 104 cases, concluded that these children "have the childlike physical qualities of their age, or they are usually dull, mentally defective, or even idiots" (Stone and Doe-Kuhlmann). A battery of psychological tests showed that both of the girls studied by Arnold Gesell were intellectually retarded, one being of

²⁹ Hoskins, *Endocrinology*, p. 58.

³⁰ Stone, Calvin P., and Doe-Kuhlmann, Lois, "Notes on the Mental Development of Children Exhibiting the Somatic Signs of Puberty Praecox," *Twenty-Seventh Yearbook, Part I*, pp. 389-397.

imbecile grade; nor did he find "any radical increase of affectivity or sociability."³¹

This evidence on precocious sexuality may be supplemented by reference to authenticated medical records of pregnancy among young girls. According to Morris Fishbein, editor of the *Journal* of the American Medical Association, a precociously developed Ukrainian girl of six, a Mohammedan girl of seven, another girl of eight years and ten months, and many girls of ten, eleven, and twelve have given birth to babies.

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The treatment of endocrine disorders

The treatment of deficiency endocrine disorders consists essentially in supplying, under medical supervision, some form of the deficient hormone, with or without related hormones, supplemented in some cases by various vitamins (such as the D or B complex vitamins). In the case of excessive functioning, a portion of the gland (especially a diseased part) and existent tumors or cysts may be surgically removed or the gland may be subjected to X-ray irradiation. The outcome of the treatment varies with the severity, type, stage, and complications of the disorder and its mode of origin—whether

³¹ Gesell, A., "Precocious Puberty and Mental Maturation," *Twenty-Seventh Yearbook*, Part I, pp. 399-409.

it is constitutional or consequent to infections, accidents, or pathological growths. The results vary from complete ineffectualness to complete restoration. Hypothyroid conditions respond better to glandular extracts than do hypopituitary disorders.

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Chapter 13

GROWTH ANOMALIES RELATED TO BIOCHEMICAL OR METABOLIC DISTURBANCES OR NERVE DEGENERATIONS

A voluminous number of writings have been produced during the present century on a considerable variety of rare and curious physical and mental disorders which appear to be clinical entities and to be caused by certain biochemical alterations or disorders of metabolism¹ due, perhaps, to some innate cellular defects. Many of the defects are attributed to the failure of a particular step in the process of metabolism which results in the deposition of lipoids (fatlike substances; *lipos*, fat; *eidos*, form) in the nerve cells and sometimes in other body cells as well. Many geneticists believe that the defects rest upon an hereditary metabolic disorder due to the absence of a specific enzyme² system traceable to a single recessive gene substitution. The occurrence of these clinical entities among some siblings and the increased prevalence of consanguineous marriages among the parents (who, however, are almost always free from the defects) have led many investigators to classify the disorders as simple Mendelian recessives. However, the ultimate explanation is still shrouded in mystery. This group of deviates, with varying proportions of

¹ Metabolism is the process by which living cells transform food materials into their own protoplasm by processes of anabolism (assimilation and regeneration) and catabolism (excretion and degeneration). A favorable balance between the two processes of building up and tearing down is essential for the maintenance of life.

² An enzyme is a ferment or compound which can produce chemical transformations of some other compound or compounds.

mental defectives among them, includes, among others, the following:

Albinism

Albinism (*albus*, white) is characterized by abnormal whiteness of the hair and skin and pink eyes, apparently due to lack of the pigment of melanin, which is related to certain aromatic compounds. The condition is often attended with photophobia, rapid, involuntary movements of the eyeballs, and astigmatism. A considerable proportion of albinos are mentally retarded in varying degrees, some being distinctly mentally defective. Albinism has been classified as a Mendelian recessive.

Pseudohypertrophic muscular dystrophy

This degenerative condition of muscle enlargement, atrophy, and weakness (see Chapter 17 on the orthopedically disabled) is accompanied by alteration of the metabolism of creatine. This element is excreted in the urine instead of being transformed and resynthesized. Some cases have been classified as sex-linked Mendelian recessives, and others as autosomal recessives.

Wilson's disease

This disease was named after S. A. Kinnier Wilson, British neurologist. This progressive disorder, first noticed during the second decade of life in children previously normal, is characterized by disease of the liver (cirrhosis) and progressive lenticular degeneration of an organ (corpus striatum) at the base of the brain in front of the thalamus. It is marked by tremors, rigidity of the trunk, limbs, and face, contractures, difficulty in swallowing and in articulation, and steadily progressive weakness, emaciation, and mental arrest. No cure has been discovered for this condition. The acute cases expire in about six months and the chronic cases in three to

seven years. The problem of the school is to obtain early diagnosis of these cases so that they may receive the discriminating care that they require. The disease has been attributed to a poison generated by the disordered liver, which has a selective action on the lenticular body; to some obscure metabolic disorder, because of the presence of silver and copper in the viscera and disturbances of pigmentation; and to a recessive gene defect, because of the noninvolvement of the parents but the occasional involvement of siblings and other relatives.

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Tredgold, *Mental Deficiency*, p. 281.

Gargoylism

This disorder was so named in 1936 by R. W. B. Ellis, W. Sheldon, and N. B. Capon of England (although it was described as early as 1908), because of the resemblance of the affected persons to the grotesquely shaped projecting water spouts in the form of men or animals—gargoyles—in mediæval architecture. This condition, which commonly is not apparent at birth, is characterized by enlargement of the spleen and liver, deposits of lipoid granules, a grotesque facial appearance (one of its marked characteristics), deformities, and, ordinarily, enlargement of the skull. Other characteristics include a saddle-shaped nose, eyes set wide apart, coarse, bushy eyebrows, a hunched back (kyphosis), short limbs, impaired motion in the joints, distended abdomen, opacities of

the cornea, and low mentality, which usually becomes apparent during the first or second year.³ Hydrocephaly is also a frequent complication. Lurie and Levy report that 50 cases have been recorded.⁴ How many cases have remained unrecognized is not known. Two of their cases had I. Q.'s of 25 and 44 (see Fig. 18). Two of Ross, Hawke, and Brown's

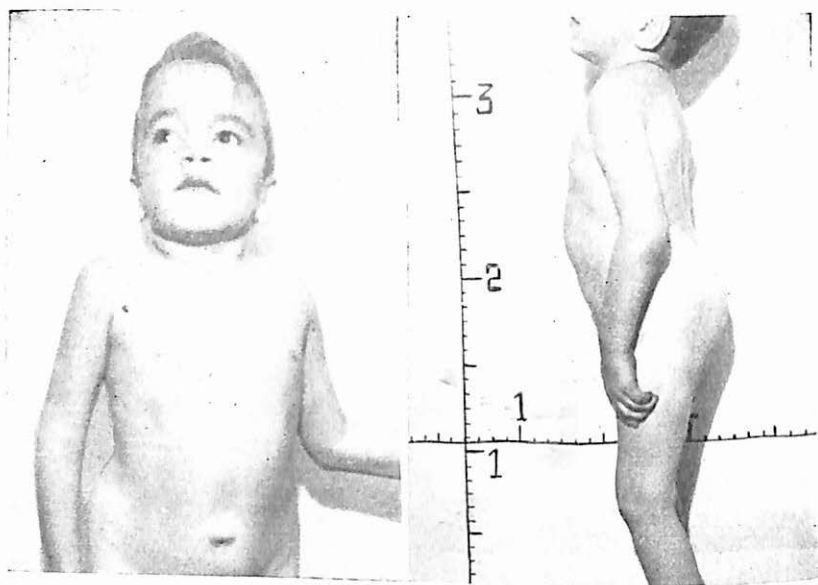


FIG. 18. C. B., a case of gargoylism, referred at 6-8 from the first grade because of lack of progress. Note the large, retracted head (circumference 22 inches), bulging forehead, large, expressionless face, depressed nasal bridge; short neck; rounded shoulders, spinal curvature, pot belly, slightly flexed elbows, barrel-shaped chest; $2\frac{1}{2}$ inches short in height but 6 pounds overweight for height and age; hearing impairment; Stanford-Binet (Form I) I. Q. 44; Vineland Social Maturity Scale S. Q. (social quotient) 71; memory very poor; spoke only a few words clearly. His brother, with an I. Q. of 25 at age 7-8, had the same malady.*

³ Halperin, S. L., and Curtis, George M., "The Genetics of Gargoylism," *American Journal of Mental Deficiency*, 1942, 46:298 f. Halperin, S. L., "Heredity and Mental Deficiency," *American Journal of Mental Deficiency*, October, 1946, 153-163 (Table XII classifies over 30 abnormalities as Mendelian recessives or dominants).

⁴ Lurie, L. A., and Levy, S., "Gargoylism: Review of Literature with Report of Two Cases," *American Journal of Medical Sciences*, February, 1944, 184-195.

* Courtesy of Louis A. Lurie and Sol Levy and the *American Journal of the Medical Sciences*, 207:184-195, February, 1944.

cases were classified as imbeciles and two as idiots.⁵ The parents invariably are unaffected, and the family histories are usually negative, although relatives and siblings have been found with the same difficulty. Instances of cousin marriages above the average incidence have been discovered in some pedigrees. Similarities have been found between this condition and amaurotic family idiocy so far as concerns the presence of certain abnormal neural conditions, such as deposits of fat in the nerve cells. The disorder has been attributed to a single recessive autosomal⁶ gene. The number of these cases that come referred from the public schools is negligible because of their infrequency and low mentality. They belong in institutions rather than in special classes, although a few of the highest mentality might be given probationary trial in a special class before being institutionalized. Many parents may well prefer to keep them at home where they can be cared for during their brief life spans.

Phenylpyruvic amentia or phenylketonuria

The latter name was suggested by Lionel S. Penrose. This type of mental defect, first identified by A. Folling in Sweden in 1934, can be definitely diagnosed by the finding of an aromatic compound (called phenylpyruvic acid; *phenyl*, a univalent radical) which has not been completely oxidized in the urine. A few drops of ferric chloride cause the urine to turn green when it contains this acid. The acid is never found in mentally normal persons. This morbid entity, which occurs in children of symmetrical body proportions, is not apparent at birth. It is marked by the development of tics, tremors, muscular hypertonicity, exaggerated deep reflexes, athetosis (slow, writhing, tentacle-like, involuntary movements, mostly affecting the fingers and hands), stereotyped mannerisms.

⁵ Ross, J. R., Hawke, W. A., and Brown, A., "Gargoylism. A Study of Four Cases," *Archives of Diseases in Childhood*, March, 1941, 71-80. Gates, *Human Genetics*, pp. 781-782.

⁶ An autosome is an ordinary chromosome other than a sex chromosome, usually paired and similar in both sexes.

apathy, irritability, and pronounced intellectual defect. Many learn to speak only a few words. Walking is often delayed until the child becomes from two and a half to eight years of age. Some, however, attain sexual maturity. Jervis discovered 161 cases among 20,300 inmates in 14 institutions for mental defectives, or 0.79 per cent, and estimates the prevalence of the disorder to be 4 cases in 100,000 of the general population (.004 per cent). Two-thirds of these institution cases were classified as idiots and one-third as imbeciles. Five per cent were offspring of consanguineous (cousin) marriages as compared with a theoretical expectation of only 0.5 per cent. Although 68 per cent showed familial incidence of the disorder, all except two resulted from the mating of two heterozygous⁷ parents, hence they were derived from a single autosomal recessive gene. To quote George A. Jervis, in a restatement of the Mendelian theory (see pp. 206 ff.):

On the hypothesis of a single recessive gene, without influence on viability, three-fourths of the sibs will be normal and one-fourth affected when both parents are heterozygous, one-half will be normal and one-half affected when one of the parents is affected and one heterozygous, no sib will exhibit the character when one parent is affected and the other is normal, and finally, all sibs will be affected when both parents are affected. In the present material, all parents of affected individuals, with the exception of one family, were normal, i.e., on the hypothesis of a single recessive gene, all but two of the recessive homozygotes⁸ are the result of the mating of two heterozygous parents.⁹

⁷ A heterozygote is an organism of mixed (or hybrid) heredity; the parents are unlike with respect to a given gene, hence the individual does not breed true with regard to the trait in question. See page 207.

⁸ A homozygote represents a pure breed, that is, the parents are alike with respect to a given gene or transmissible character, hence the individual breeds true in regard to that character. See page 207.

⁹ Jervis, George A., "A Contribution to the Study of the Influence of Heredity on Mental Deficiency: The Genetics of Phenylpyruvic Oligophrenia," *Journal of Psycho-Asthenics*, 1939, 44, 2:13-24; also "Inherited Biochemical Alterations in Certain Types of Mental Deficiency," *ibid.*, 1937, 42, 2:101-116 (references, including a tabulation of recorded cases of amaurotic family idiocy and Wilson's disease).

The most recent study of the disorder is by Frazier of ten cases, six males and four females, ages nine to fifty-eight, among 3,000 inmates in the Orient State School in Ohio, with an I. Q. range from 9 to 19. In finding grounds for accepting the Mendelian recessive interpretation, he offers the following comments. Though "there is little social importance in the condition because of its rarity . . . it is, of course, extremely important in families in which it has appeared; the low intelligence, apathy, and irritability make a difficult situation in the home, and the mental hygiene of the family usually requires early institutionalization. Further, sexual maturity is reached and there is on record a case of pregnancy. . . . Marriage to a cousin, or marriage into another family where this condition is known to exist, is strongly contraindicated because the frequency of the carrier state in relatives is tremendously increased over the general population."¹⁰ Because of their low mental status, few, if any, of these infrequent cases will reach the public schools.

The condition, apparently, is caused by the hereditary lack of some form of amino acid metabolism.

Niemann-Pick's disease

(First identified in 1914 by Albert Niemann and Ludwig Pick in Germany.) This rapidly progressive familial disorder of development, largely confined to Jewish children, begins in early life, often at birth, and usually ends fatally within the first two years. The outstanding features include enlargement of the liver and spleen, anemia, a brownish skin, the infiltration of lipid fat in the form of phosphatide in the large interstitial cells in every tissue, and arrest of mental and physical development. It is occasionally associated with amaurotic idiocy, showing the cherry-red retinal spot characteristic of that disorder. Perversion of lipid metabolism is the usual

¹⁰ Frazier, Robert L., "Phenylpyruvic Amentia," *American Journal of Mental Deficiency*, April, 1947, 577-586. See also Gates, *Human Genetics*, pp. 1088-1091.

explanation. Reeves and Anderson have traced 48 cases in the medical writings.¹¹ Of course, many cases have never been correctly diagnosed. Because of their short life span, these children do not create any problems for the schools so far as educational adjustment is concerned.

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Amaurotic idiocy

Four types or variations of this strange nervous disorder have been recognized, all apparently produced by the same pathological process (lipoidosis or disturbed lipid metabolism) and all manifesting a common triad of progressive mental impairment, muscular anergia or asthenia (weakness), and loss of vision.

The infantile form (Tay-Sachs disease). This form, the first to be identified (by Warren Tay in England in 1881, who described the ocular changes, and by Bernard Sachs in New York City in 1887,¹² who discussed the brain condition), originates during the first few months of life, usually during the first six months, in apparently normal and healthy infants, ordinarily of Jewish extraction. There is no racial predilection in the later forms. The process of nerve cell and retinal degeneration proceeds very rapidly, with the result that the infant soon becomes totally blind through optic atrophy, totally incapacitated muscularly, and severely deteriorated mentally (profoundly idiotic). A fatal termination follows a

¹¹ Reeves, David L., and Anderson, Lucile R., "Niemann-Pick's Disease Associated with Amaurotic Idiocy," *Bulletin of the Los Angeles Neurological Society*, December, 1941, 177-190.

¹² Sachs, Bernard, "On Arrested Cerebral Development with Special Reference to Its Cortical Pathology," *Journal of Nervous and Mental Diseases*, 1887, 14:541-553.

year or two after the onset and before the end of the third year. The age of onset of 70 cases from 27 sibships summarized by Slome was from one month to twelve months, and the age of death was from seven to thirty months.

Examination of the retina reveals in many cases a cherry-red spot in a light gray oval patch at the center of vision (called the *macula lutea*). This red spot, absent in the later forms, is regarded as a pathognomonic (specifically characteristic) sign of the infantile type. The varied muscular involvements in different stages include muscular twitching, clonic movements (spasms of contraction and relaxation), spasticity of the extremities, and growing muscular weakness and emaciation. Eventually the child is unable to support the head, sit up, turn over in bed, or swallow, and dies of extreme marasmus and inanition. Although the child can distinguish only between light and darkness, hearing remains largely intact. Mentally he becomes increasingly unobservant and apathetic. Two Jewish siblings, less than three years of age, carried in the parents' arms into the writer's clinic in the University of Pittsburgh in 1913, were unable to stand, sit, talk, see, or respond to any of the psychological tests except that they seemed to give a weak motor response to loud noises. They were leading a purely vegetative existence.

Marburg attributes the asthenia characteristic of the anomaly to an endocrine deficiency, the lack of functioning of the adrenal medulla, the internal part of the gland which secretes epinephrine.¹³

Almost all parts of the nervous system, but especially the brain, show pathological changes, most important of which are infiltration of the nerve cells with granules of lipoids, swelling of the cytoplasm of the cells (the protoplasm surrounding the nucleus), and destruction of the neurones.

Family history investigations have revealed the incidence of the condition in several generations of the affected families

¹³ Marburg, Otto, "The Endocrine Glands in Infantile Amaurotic Idiocy," *Journal of Nervous and Mental Disease*, November, 1944, 450-461.

and a high incidence of consanguineous marriages, although the parents themselves are normal. Hoben, for example, reports 15 per cent of the parents to be first cousins. Consanguinity, reputedly the most important identifiable factor, affords greater opportunity for the operation of the genetic recessive mechanism supposedly at the base of the disorder.

No medical treatment or cure exists for amaurotic idiocy: all forms invariably prove fatal. Although genetically all forms are of great significance, the infantile and delayed varieties are of little or no significance to the educator or social worker, except as they may cause difficulties in the home which may create problems for society and for the schools.

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Late infantile form (G. Jansky). This variant goes through a slow course of mental and physical deterioration, with increasing muscular rigidity (decerebrate rigidity) from the onset at about the third or fourth year to its fatal termination at about the end of the fourth year. These children create serious problems of care and protection for the home or institution for a year or more.

Juvenile form (first identified by W. Spielmeier and H. Vogt). Children afflicted with this variant usually develop normally mentally and physically until about the age of five to seven, when failure of vision and mental deterioration become

increasingly apparent, both progressing slowly until death supervenes at from fourteen to eighteen. Of 83 cases from 32 sibships studied by Sjögren in Sweden, the age of onset varied from two to eleven years and the age of death from nine to twenty-four.¹⁴ Genetic data indicated to him that the abnormality is the result of the action of an autosomal recessive gene. Other symptoms that may develop during the course of the degenerative process include epileptic seizures (in about 80 per cent of cases), usually appearing at from ten to twelve, psychotic manifestations—confusion, delusions, and hallucinations¹⁵—and general spasticity. With the growing muscular tonicity and spasticity, increasing flexion of the arms and legs and walking difficulties develop. The child begins to stumble and stoop forward more and more as he walks. The optic atrophy is less pronounced in this form and the cherry-red spot is frequently replaced by a brownish pigmentation of the retina (retinitis pigmentosa). The isolated cases that infrequently reach the schools do not cause any special problems until the visual, mental, and neuromuscular changes become apparent. But problems may arise from the fact that an isolated case may not be recognized for what it is and therefore no attempts, or only bungling attempts, will be made to adjust the educational procedures to the child's peculiar requirements.

The adult form. This form develops considerably later than the juvenile variant, often in the late twenties, and is marked by slowly developing dementia, and frequently by the deposits of pigment in the macular region of the retina; but muscular rigidity is usually absent. These cases, fortunately rare, obviously constitute serious problems in the homes and are of direct concern to the educator as soon as the deterioration becomes patent.

¹⁴ Sjögren, T., "Die Juvenile Amaurotische Idiote," *Hereditas*, 1931, 14:197.

¹⁵ Delusions are false judgments or beliefs that are given credence in spite of the external evidence of their falsity. Hallucinations represent subjective perceptions of objects in the absence of objective stimuli.

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Four rare neuromuscular disorders

Thumbnail descriptions, with references, are given of four degenerative neuromuscular disorders afflicting children, often hereditary and sometimes associated with mental deficiency.

Gaucher's disease (primary congenital form): described by Phillippe C. E. Gaucher, French physician, in 1882; diagnosed from finding Gaucher cells, large, pale reticular cells containing a lipoid substance, kersasin (shown by Emil Epstein), es-

pecially in the spleen, lymph nodes, and bone marrow; enlarged spleen and liver, except in osseous type; splenic anemia, tendency toward bleeding, sometimes skin pigmentation; occasionally neurological symptoms from degeneration of cortical neurones, restricted to children before six months, including arrest of physical and mental development, spastic extremities, elbows and legs bent (flexed), sometimes idiocy; course very rapid in infants under one, with fatal termination; slower in older children and adults; no racial predisposition; females predominantly affected; many symptoms vary with age of onset; explained as congenital disturbance of lipid metabolism or as a Mendelian recessive.

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Hand-Schüller-Christian disease: described by Alfred Hand, of Philadelphia, in 1891, Henry A. Christian, of Boston in 1919, and Arthur Schüller, of Vienna in 1916; a disturbance of cholesterol metabolism; diagnosed on the basis of cholesterol in large reticular "foam cells" throughout the body but especially in the membranous bones, particularly in the skull; protrusion of eyeballs (exophthalmos); kidney disease with excess of urine but not of sugar (diabetes insipidus); yellowish-brown skin lesions; gingivitis, loss of teeth; sometimes dwarfism, infantilism, and mental retardation; slow chronic course; 34 of 50 recorded cases began in first decade, may continue into third decade; males predominate, but no race preference; no hereditary basis, according to Davison, although sometimes classed as a recessive; treatment (low fat diet, insulin, deep X-ray) only moderately successful.¹⁶

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Levine and Solis-Cohen, "Gaucher's Disease."

Pack and Silverstone, "Gaucher's Disease, Report of a Case."

Shands and Raney, *Handbook of Orthopedic Surgery*, p. 79.

Diffuse sclerosis or Schilder's disease: first classed as a distinct entity by Paul Schilder, Vienna neurologist, in 1912; a form of brain degeneration various types of which have been recognized, although clinical differentiation among them is often difficult; characterized by extensive degeneration of the

¹⁶ Davison, "Degenerative Diseases," in *Practice of Medicine*, pp. 530-535.

white matter of the central hemisphere involving mostly the myelin sheaths (demyelination) and, to a lesser extent, the axis cylinders (central conduction part of the nerves). As a result of myelin destruction, fat is formed and at a later stage extensive scars develop (sclerosis) involving large areas of the subcortical white matter. The cortex remains intact. Clinical manifestations vary: spastic paralysis of one or more extremities, speech disorders affecting comprehension of speech or articulation, loss of memory and apathy are among the most commonly observed symptoms; disturbances of vision leading to blindness are observed when the process involves the occipital lobes; headache, vomiting, and convulsions are often present in the acute forms, whereas in the chronic cases psychic disorders (visual and auditory hallucinations, impulsive laughter, paranoid trends) may prevail. Ultimately, physical, emotional, and intellectual decay become outstanding. Over half of the cases of diffuse sclerosis originate before the age of fourteen. Acute forms may prove fatal within a few months; the chronic form may continue up to ten or twenty years. The cause of the disease has not been established as yet. Some types may be due to a recessive genetic mechanism. There is no treatment. Children who made normal school progress prior to the onset may have to be transferred to special classes if they are able to continue after deterioration sets in.

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Friedreich's ataxia or hereditary ataxia: named after Nicholas Friedreich, Heidelberg physician; an heredofamilial degeneration, apparently recessively determined, of the posterior columns of the spinal cord, pyramidal tract (nerve fibers to the motor ganglia and to the cord), and cerebellum, usually beginning before the age of fifteen, often affecting more than one member of the family, more frequently males, progressing very slowly into a state of helplessness; one of earliest signs is muscular incoordination, beginning in the lower limbs and proceeding upward, producing serious ataxia (unsteady balance, unsteady, waddling, swaying gait from involvement of the cerebellum and/or loss of muscle sense, with jerky placement of widely separated feet); the child stumbles and falls, arm and finger movements become clumsy, handwriting becomes scrawly, speech slow, jerky, indistinct, and sometimes scanning; spine often curved (thoracic scoliosis); loss of ankle and knee reflexes; hyperextension of the big toe; eventually paralysis, contractures, and wasting; rapid, involuntary movements of the eyeballs when eyes are moved to side; sometimes optic atrophy and impaired vision; no mental impairment in many; others deteriorate, some considerably; no specific medical treatment. The child can continue in school until the muscular disabilities become too severe. Special facilities should be provided in special classes or otherwise, including muscle exercise to retard the advance of the symptoms and orthopedic care to prevent deformities.

The challenge of preventive programs

The prognosis of all of the special types treated in this chapter is very unpromising from the standpoint of both prevention and cure. The discovery of effective methods of prevention, the greatest desideratum, will constitute a crowning achievement in the field of genetic research, which will doubtless be reflected by parallel advances in wider areas. A large body of information has been accumulated during the past 60 years by the numerous biochemical, histological, and genetic

investigations inspired by the presence in society of children afflicted with these baffling morbidities. But the knowledge thus far available is inadequate for mapping effective and practical medical, sociological, educational, or eugenic programs.

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Chapter 14

EPILEPTIC SEIZURES IN CHILDREN¹

A major, poorly comprehended nervous malady

Epilepsy, which is essentially a malady of childhood and adolescence, is one of the most tragic disorders to which the human nervous system is subject, although the seizures themselves are brief and painless. Many of the victims of this protean nervous disturbance would be all right if they could be freed from their paroxysmal attacks. Relieved of the seizures, they would be able to lead a normal social and economic existence free from the misunderstanding, pampering, and overprotection, or discrimination, ridicule, social isolation, segregation, and disdainful treatment of which epileptics sometimes become innocent victims. The disease, known in ancient times as the sacred malady (*morbus sacer* and *morbus divinus*—because it was thought to be caused by some god, a reminder of the theory of demon possession), the falling disease (*morbus caducus*), or the slobbering disease (*morbus incontinentis*), and later commonly referred to as epilepsy (from *epilepsis*, seizure), has remained throughout the ages one of the great medical conundrums. Perhaps more progress has been made in treating the condition since the new anticonvulsant drugs were devised and in unraveling its mystery since the techniques of pneumoencephalography and electroencephalography were devised than was achieved in all the preceding centuries. But in spite of the thousands of articles, mono-

¹ Appreciation is due Hans Löwenbach, M.D., Associative Professor of Neuropsychiatry, Duke University, for reading this chapter. William G. Lennox, M.D., Assistant Professor of Neurology, Harvard University, has checked various items of information in correspondence.

graphs, and texts written on the subject by some of the world's greatest medical luminaries (Hippocrates, the "father of medicine," Galen, Thomas Willis, Bravais, Hughlings Jackson, Brown-Sequard, and Gowers) and in spite of the great progress recorded during the last 10 or 15 years, the essential neural and physiochemical nature of epileptic phenomena, of which a great variety exists, still remains shrouded in considerable obscurity.

Nonepileptic convulsive nervous attacks

Infants and young children, especially of the spasmophilic diathesis (abnormal sensitivity to stimulation, often shown in a low convulsive threshold manifested in spasms), readily develop a variety of so-called fits, spasms, convulsions, or seizures. These may be precipitated by gastrointestinal upsets, the strain of teething, worms, temporary cerebral congestion, acute infections, toxemias, or lack of calcium in the blood from inadequate parathyroid secretion (cases of tetany). The large majority of these convulsions are transient phenomena, and, although often indistinguishable from true epileptic seizures, do not constitute true epilepsy. These spasms, referred to as eclampsic by F. R. P. Taylor and as spasmophilic by some other writers, ordinarily cease by the second or third year spontaneously or as a result of proper treatment (diet, sunlight, cod liver oil, and calcium bromide). Thom found that about 7 per cent of Boston children had one or more convulsions during the first five years of life, only a few of which were epileptic.² Of 2,500 children less than seventeen years of age subject to convulsions studied in "private office and in hospital practice over a period of 20 years," Peterman ascribed 33.4 per cent to acute infections, 26.3 per cent to idiopathic epilepsy, 14.2 per cent to cerebral birth injuries, and 26.1 per cent to other causes. The large majority of the convulsions developing during the first month were attributed to birth

²Thom, Douglas A., "Convulsions of Early Life and Their Relation to the Chronic Convulsive Disorders and Mental Defects," *American Journal of Psychiatry*, January, 1942, 570 f.

injuries, and those developing between the first month and the thirty-sixth month, to acute infections. Cases of idiopathic epilepsy constitute by far the largest single group in the age period from three to nine years inclusive, and 73.2 per cent of all the cases in the age group from ten to sixteen.³ Leonard Guthrie has estimated that only about 10 per cent of infantile convulsions are or become true epilepsies. Many pediatricians regard the convulsions induced in childhood by fevers as merely the equivalent of a chill in an adult, and believe that nonepileptic infantile convulsions are relatively innocuous and leave no permanent injury, even when frequent, although the child may appear dull for a time. This view is questioned by Lennox,⁴ who finds that approximately 20 per cent of those who developed seizures at puberty or later were subject to isolated infantile convulsions.

Distinctions between epileptic and hysterical convulsions. Epileptic and hysterical convulsions are often confused on the basis of certain superficial resemblances. The following diagnostic differentials may prove helpful in preventing the confusion.

(1) Epileptic convulsions are, in the large, involuntary and uncontrollable, whereas the hysterical ordinarily are staged. Hysterical convulsions are bids for sympathy and usually do not occur in the absence of an audience. It should be recognized, however, that a few epileptics can and do "throw" convulsions voluntarily that are almost indistinguishable from the genuine ones. They may do this in order to have their way or as a means of escape from disagreeable experiences.

(2) The hysterical patient takes pains to avoid suffering injury in falling, although the lips are sometimes bitten; but the victim of grand mal makes no such effort, except perhaps in the early prodromal stage.

³ Peterman, Mynie G., "Convulsions in Childhood," *American Journal of Diseases of Children*, October, 1946, 399-410.

⁴ Lennox, William G., *Science and Seizures* (2d edition). New York: Harper & Brothers, 1946, p. 96.

(3) The prodromal period lasts longer in hysterical convulsions, but the aura is absent or less obvious.

(4) Consciousness is less deeply affected in an hysterical attack.

(5) The "hysterical facies" (emotional utterances, or sobbing) are absent in epilepsy.

(6) The pupils are not expanded as in grand mal convulsions, when the latter are accompanied by profound unconsciousness.

(7) Some of the convulsive movements in hysteria are different, such as grasping and striking at persons and things.

(8) The brain waves characteristic of epilepsy are lacking in hysteria. The brain wave technique apparently enables the epileptologist to make an accurate differential diagnosis in doubtful cases. Epileptic attacks apparently result from, or accompany, cerebral dysrhythmia, whereas hysterical attacks are provoked by mental or emotional conflicts, and not by a disordered rhythm of the electrocortical waves.

IDIOPATHIC AND SYMPTOMATIC TYPES OF EPILEPSY

Although epileptic attacks tend to follow the same pattern for any given individual, no two individuals have exactly the same kind of seizures. In fact, the sensory, motor, emotional, and intellectual manifestations differ so greatly in different epileptics so far as concerns the mode of onset, appearance, severity, and aftereffects that some authorities tend to refer to the condition in the plural rather than in the singular. They prefer to speak of the epilepsies rather than of epilepsy as a nosological entity. They regard epilepsy as a generic concept for a combination of symptoms—a symptom-complex—and not as a definite disease entity with a definite, invariable causation.

Basic genetic types

The epilepsies are classifiable into two major categories, idiopathic and symptomatic. In the case of idiopathic epi-

lepsy (also referred to as true, essential, or cryptogenic) no definite causative factor is discoverable other than a constitutional predisposition (diathesis) or hereditary tendency toward nervous instability, or toward paroxysmal seizures or, to use the latest concept, toward certain patterns of electrical brain waves, or toward a disturbance of the rhythm-regulating mechanism of the cerebrum (called cerebral dysrhythmia by Lennox).⁵ The exact cause is obscure or unknown, hence cryptogenic (*cryptos*, hidden); or the disorder is self-generated, hence idiopathic (from *idios*, one's own). Many of the exciting or contributing factors that may precipitate convulsions in persons with a low convulsive threshold are well known: infections—from meningitis or encephalitis—toxemias, gastrointestinal upsets, the stresses of teething, puberty, and pregnancy, disturbance of the chemical balance (from an excess of insulin, low blood sugar, or uremic poisoning), alcohol, lead, and other poisons, and emotional upsets.

The symptomatic, or traumatic, or acquired group can sometimes be traced to definite antenatal, natal, or postnatal brain injuries or lesions affecting the motor cortical areas. The brain lesion may represent a developmental anomaly; or lesions or injuries may be the result of a mechanical injury, infection, anoxia, hemorrhage, or blocked circulation. The cerebral injuries may result in tumors, scars, cysts, abscesses, adhesions, or areas of hardening (sclerosis or degenerative atrophy). Local reflex irritations from injured areas may provoke attacks in the predisposed. In old age, convulsions are associated with arteriosclerosis. Lesions in the motor cortex, however, do not always produce convulsions. There is scarcely a cerebral lesion that may not produce seizures, but no lesion can be depended upon to do so invariably. Lesions are more likely to produce epilepsy in spasmophilic persons or in persons with a low innate convulsion threshold.

Many attempts have been made experimentally and from

⁵ *Ibid.*, p. 19.

studies of brain pathology to determine the specific exogenous factors that are responsible for convulsions or that are connected with the epileptic diathesis or its basic pathology. Many explanations have been propounded, most of which are mere hypotheses still in need of confirmation. Among such factors may be mentioned: metabolic disorders;⁶ deficiency of sugar in the blood (hypoglycemia); increase in the white blood corpuscles;⁷ excess of fluid in the brain, producing an increased permeability in the cell surfaces;⁸ deficient oxygen content in the blood (anoxemia), caused by some vascular or metabolic disturbance, which increases the excitability of the neurones;⁹ decreased oxygen tension;¹⁰ disturbances (storms) in the vegetative centers of the nervous system;¹¹ sclerosis in the cerebellum and in the middle horn of the lateral ventricle (hippocampus), "vasospasms" being the important basis of seizures;¹² and agenesis (lack of development) of the frontal lobe.¹³ The psychogenic theories have emphasized the role of disturbed emotions as the precipitating cause of seizures, and the states of unconsciousness induced by the attacks have been interpreted as escape mechanisms, or means for dodging disagreeable experiences or for retreating from the harsh realities

⁶ Collier, J., "Lumlein Lectures on Epilepsy," *Lancet*, March 24, 1928, 1:587-591.

⁷ Guirdham, A., and Pettit, A. W., "The Hematology of Convulsions," *Journal of Mental Science*, July, 1936, 82:371-393.

⁸ Spiegel, Ernest A., and Spiegel-Adolf, Mona, "Fundamental Effects of Epileptogenous Agents upon the Central Nervous System," *American Journal of Psychiatry*, March, 1936, 92:1145-1168.

⁹ Lennox, William G., and Gibbs, Erna L., "Oxygen Saturation of the Arterial Blood in Epilepsy," *Archives of Neurology and Psychiatry*, June, 1936, 35:1198-1202.

¹⁰ Lennox, W. G., and Behnke, Albert R., "Effect of Increased Oxygen Pressure on Seizures of Epilepsy," *Archives of Neurology and Psychiatry*, 1936, 35:782-788.

¹¹ Yakolev, Paul I., "Neurologic Mechanism Concerned in Epileptic Seizures," *Archives of Neurology and Psychiatry*, March, 1937, 37:523-554.

¹² Spielmeyer, W., "The Anatomic Substratum of the Convulsive State," *Archives of Neurology and Psychiatry*, May, 1930, 23:869-875.

¹³ Bateman, J. Fremont, "Cerebral Frontal Agenesis in Association with Epilepsy," *Archives of Neurology and Psychiatry*, September, 1936, 36:578-585.

of life.¹⁴ The attempt to find a single cause for all kinds of epileptic phenomena is probably foredoomed to failure.

Types of convulsions

Grand mal (the great malady). In about half the cases the grand mal attack (and, less conspicuously, some petit mal also) is preceded by certain premonitory symptoms or warnings in the prodromal stage. These may consist of feelings of discomfort or depression, temperamental changes (moodiness, irritability), tingling, numbness, facial changes, or twitchings; and the aura (Greek for breeze or breath), the stage that immediately precedes the convulsion and merges with it.¹⁵ The sensory symptoms of the aura may consist of flashes of light, sights, sounds, tastes, and epigastric feelings (nausea). The motor symptoms include movements of certain muscles, tremors, eyewinks, and coughing. The psychic signs may consist of sudden ideas, recollections, illusions, or hallucinatory images. Because the convulsion originates in the brain, the notation of the external symptoms may be of value in indicating the brain areas in which the convulsive impulse originates.

The aura is immediately followed by a sudden loss of consciousness, one of the cardinal features of the attack, without apparent cause (such as cardiac failure or asphyxia), and the victim falls as if suddenly struck dead. The direction of the fall is determined by the order in which the muscles become activated. The spasm begins with tonic cramps or a condition of tetanic rigidity. That is, the muscles contract violently and are held rigid temporarily. One limb may grow

¹⁴ Carlisle, Chester L., "The Etiology of Idiopathic (Nonorganic) Epilepsy," *United States Veterans' Bureau Medical Bulletin*, March, 1929, 5:161-173; Clark, L. Pierce, "Psychology of Essential Epilepsy," *Journal of Nervous and Mental Disease*, 1926, 63:575-585; Fremont-Smith, Frank, "The Influence of Emotion in Precipitating Convulsions," *American Journal of Psychiatry*, January, 1934, 13:717-723; Hamill, Ralph C., "Petit Mal in Children," *American Journal of Psychiatry*, September, 1936, 93:303-312.

¹⁵ Some writers draw no distinction between the prodromal stage and the aura.

stiff after the other, and the head and eyes may turn. The vigorous contraction of the chest muscles may cause a guttural cry, gurgling, groaning, or a sucking sound and may inhibit the breathing and cause cyanosis (blueness of the skin). The pupils become dilated. This stage, which may last for as long as a few seconds or one or two minutes, is succeeded by violent clonic contractions and relaxations, lasting from one to two or three minutes. This is a stage of intense, not to say dramatic, discharge of energy. Many muscle groups are thrown into vigorous, wild, incoordinate contractions and relaxations. The legs and arms jerk and writhe wildly; the facial muscles may present a picture of vigorous contortions, with possible grinding of the teeth and biting of the tongue; the heaving chest may produce violent movements of inhalation and exhalation with expulsion of saliva; and the whole body may be bathed in profuse perspiration. The eruption occasionally is accompanied by the loss of control of the bladder or the bowels. The movements gradually grow slower and weaker and the explosion terminates in a state of painless relaxation or comatose condition characterized by deep stertorous breathing (stertor), which may merge with natural sleep. Upon awakening from the deep sleep after varying lengths of time, the patient may feel exhausted, he may suffer from sore muscles, headaches, and spells of vomiting, and he may remain mentally dull and confused for varying lengths of time. These automatic and uncontrollable episodes differ greatly from patient to patient and from time to time for the same patient in severity, extensiveness of motor involvement, duration, and aftereffects, but they rarely prove fatal no matter how severe the attack may be. When death supervenes it is commonly caused by a brain injury from a severe fall, from asphyxia at night from rolling over and smothering, or from heart failure.

William Spratling found that the duration of the total seizures for 516 cases varied from half a minute to five minutes with an average of 1.7 minutes. Forty per cent of 1,500 pa-

tients reported by Lennox¹⁶ had convulsions either day or night; 36 per cent had only diurnal and 15 per cent only nocturnal attacks. Some changed from the one to the other. Of course, many nocturnal seizures go unrecorded.

Petit mal (the small illness; pronounced petty mahl). In this form of attack, which usually comes on without warning or aura, the patient suddenly suffers a momentary lapse of consciousness, lasting from 5 to 30 seconds. Although the patient rarely falls, he becomes suddenly dazed, and may drop the object he holds. He may grow pale, display a fixed stare, and evince slight twitchings of the eyebrows or eyelids and, less frequently, of the shoulders and arms. He may continue his activity or, more frequently, stop and resume it when consciousness returns abruptly, usually with an awareness of his "absence" or the blackout of his consciousness. The *petit mal* attacks occur far more frequently than the *grand mal*—sometimes several hundred a day—but they may have remained unnoticed for years because of their mild and transitory nature. This type of seizure is predominantly a childhood and adolescent disorder; but, though it tends to disappear as adulthood is reached, it may, if unchecked by proper treatment, persist throughout life.

DISTINCTION BETWEEN PETIT MAL AND ALLIED SEIZURES. Care must be taken not to confuse the *petit mal* seizures with the following attacks.

(a) Syncope or fainting spells, which may be induced by excessive heat, the sight of blood, a gruesome encounter, the receipt of distressing news, or long standing at attention, and which are usually preceded by feelings of weakness, nausea, and dizziness. The patient blanches, perspires, and falls to the floor. In the recumbent position the blood pressure suffices to restore the circulation.

(b) Myoclonic shocks, or quick, clonic jerks of the arm and trunk muscles without the loss of consciousness.

¹⁶ Lennox, *Science and Seizures*, p. 39. It is not apparent whether the *petit* *mals* are included.

(c) Akinetic seizures, which are sudden attacks of head nodding and limpness in which the subject may fall but get right up again. These seizures, in which consciousness often is retained, sometimes occur scores of times a day.

(d) Pyknolepsy, which consists of very frequently recurring (or aggregated) mild epileptiform attacks affecting children between the ages of four and twelve. They may occur almost daily for weeks, months, or years without interfering with normal mental development and eventually cease spontaneously (William J. Adie, 1924). Since the electric brain waves and the clinical symptoms are identical with those of petit mal,¹⁷ these attacks may be merely a particular pattern of petit mal. Lennox indeed uses the term pyknolepsy as a synonym for the classical form of petit mal.

(e) Petit mal variants, associated with cerebral lesions, which are far less frequent than petit mal, and which display a different pattern of dysrhythmia, as shown in Fig. 19.

Focal or Jacksonian spasms (from Hughlings Jackson). In these attacks in which the subject remains conscious and can witness the automatic and uncontrollable performance and feel the accompanying numbness, the twitchings begin in one arm, hand, leg, foot, or one side of the face or one muscle group. Occasionally the spasm may spread to other muscle groups on the same side; or they may engulf the whole body and terminate in a generalized grand mal convulsion with loss of consciousness. This type of convulsive disorder points to a definitely localized (focal) brain lesion. The particular seat of the lesion is indicated by the portion of the body in which the contractions originate. Once when the writer was administering a psychomotor test to an epileptic in the New Jersey State Village for Epileptics, the examinee interrupted his performance while he observed his left hand which suddenly began to twitch. The right side of his head revealed a depressed area over the motor hand region where he had been kicked by a mule some years before.

¹⁷ Lennox, *Science and Seizures*, p. 27.

Serial attacks (or status convulsus, or status epilepticus). These severe intermittent spasms, which may begin as focal attacks and then become generalized, occur so rapidly that the patient does not regain consciousness between the attacks. The condition has been known to continue for days with hundreds of seizures each day and with fatal consequences due to exhaustion or to intercurrent pneumonia. The usual course is a gradual recovery of consciousness, followed by a period of confusion and great exhaustion, with occasional postepileptic paralysis or even psychosis. The old treatment, placing the patient in a mustard pack or giving him a continuous hot bath, ordinarily interrupted the series, although I have witnessed patients succumb in the bathtub. Fortunately, this condition can now be arrested promptly in most cases with the use of the modern anticonvulsant drugs.

Epileptic equivalents, psychic or psychomotor attacks, or somnambulistic epilepsy. This rare and ill-defined condition, which may precede, follow, or take the place of (hence "epileptic equivalent") the convulsive seizures, is characterized by automatic performances (automatisms) in a twilight state of consciousness with subsequent forgetfulness (amnesic episodes) of all occurrences. During this state of clouded consciousness, the subject may walk about as if in a somnambulistic state, muttering incoherently and performing various purposeless acts. Or he may perform complicated activities as if he were conscious of what he was doing without any recollection of the events when normal consciousness may suddenly return. He may wander away from the institution and find himself in a distant city where he may have obtained a job under an assumed name. Often irritable and morose rather than hilarious, he may suddenly give vent to his feelings in a torrent of abusive language and engage in general roughhouse. He may become violent when resisted, and may commit crimes of aggression, even homicide, with or without any provocation and without subsequent recollection. Mild tonic cramps may sometimes occur—a clamping

of the jaws, stiffening of the arms and legs, drooling, or a darkening of the face—unaccompanied by falling. Sometimes only unaccountable alterations of disposition occur, such as violent outbreaks of impulsiveness or assaultiveness (“epileptic furor”). Some conduct disorders are probably epileptic equivalents precipitated by brain disturbances, as shown by the epileptiform character of the brain waves. These episodes differ greatly in duration, from a few minutes to a few hours or several days.

*Differentiation of the different types
of attacks by the pneumoencephalograph
and the electroencephalograph*

The greatest contributions made by modern neurological research are the demonstrations by means of the pneumoencephalogram (described on p. 253 f.) of the presence of atrophies or destroyed brain areas in the brain-injured cases, the great magnification and visible transcription of the electrical impulses of the cerebral cortex by means of the electroencephalograph (described on p. 256) and the correlation of different brain wave patterns with different kinds of epileptic seizures. These techniques supply improved tools for investigating the brain mechanisms of the epilepsies.

In making electric encephalograms the subject reposes or sits quietly with eyes closed in a room wired to exclude vagrant electrical or radioactive waves. Electrodes are attached to the right and left frontal, parietal, and occipital regions of the skull. Different areas of the cortex generate slightly different waves. Records may be taken with the eyes open while the subject engages in some limited mental or physical activity, or during periods of heavy breathing. Hyperventilation (heavy breathing) tends to elicit the epileptic brain patterns in persons with an epileptic diathesis.

Abnormal waves (dysrhythmia) may be too fast or too slow, or too large or too small, and may assume unusual patterns. In grand mal attacks, the waves are of high voltage and are

faster than normal; in petit mal seizures, they are of high voltage and are alternately fast and slow (three a second), with a dart and dome effect; in the petit mal variant, they are of lower voltage with a blunt spike and wave effect (two per second); in psychomotor attacks the waves are of the high voltage slow type, some of them square-topped (see Fig. 19). Large, slow waves spring from areas of localized lesions. During the paroxysm the waves are greatly increased in amplitude and rate. In intervals between attacks, transient dysrhythmias may occur although no outward evidence appears of the "subclinical," or "larval," or inward disturbances. Premonitory wave disturbances often herald the approach of

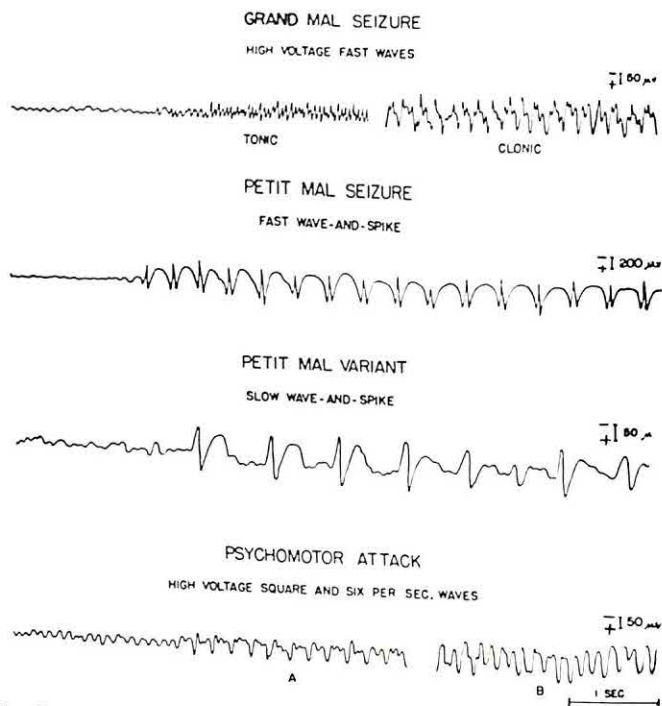


FIG. 19. Samples of brain waves or brain dysrhythmia (electroencephalograms) in grand mal, petit mal, petit mal variants, and psychomotor attacks.* The patient's normal brain rhythms are at the left of each tracing. A time interval of a second is shown by the line at the bottom.

* After William G. Lennox.

a brain storm. A series of fast waves almost always precedes the grand mal attacks. The waves may spread from a localized lesion or they may arise in many areas coincidentally. The waves can be slowed by some anticonvulsant drugs, for instance, phenobarbital. Sometimes the waves may be a mixture of two types. The wave patterns are characteristic for a given individual.¹⁸

In a study of institutionalized epileptics, feeble-minded, most subject to grand mal convulsions, a marked incidence was found of very slow brain waves (about 25 per cent of the 300 investigated) and of psychomotor forms of attack (26 per cent), compared with noninstitutional cases. Psychomotor epilepsy was considered to be at the basis of the difficult behavior cases. "Personality disorder was the real reason for commitment rather than actual seizures."¹⁹

In a study of 873 electroencephalograms, the "highest per cent of EEG abnormalities was noted in the groups with the diagnosis of epilepsy." Fifty per cent of 469 admitted and 70 per cent of 206 discharged with the diagnosis of epilepsy had abnormal encephalograms.²⁰ Not all epileptics show abnormal brain waves and not all with abnormal waves are subject to seizures.

In a third extensive investigation based on 369 cases in an institution for epileptics (243 idiopathic and 126 symptomatic cases), ages five to eighty, fewer abnormal waves were found among the symptomatic than among the idiopathic inmates. Wave and spike forms were more characteristic of the idiopathic type, and unilateral asymmetries and generalized and isolated spikes among the symptomatic cases. The wave

¹⁸ For a description of the brain waves that are connected with the other forms of seizures see Gibbs, Frederic A., Gibbs, Erna L., and Lennox, William G., "Electroencephalographic Classification of Epileptic Patients and Control Subjects," *Archives of Neurology and Psychiatry*, August, 1943, 111 f.

¹⁹ Belinson, Louis, and Cowie, William S., "Electroencephalographic Characteristics of Institutionalized Epileptics," *American Journal of Mental Deficiency*, July, 1947, 9-15.

²⁰ Rossen, R., "A Critical Analysis Obtained from 873 Electroencephalographic Examinations," *Navy Medical Bulletin*, November, 1947, 494-503.

and spike forms were significantly most prevalent among the younger patients up to age twenty-five. After that age the incidence of this form was negligible in the symptomatic group, but appreciable (although waning) in the idiopathic group.²¹

*Relative prevalence of the major kinds
of epileptic phenomena*

The following statistics, only "roughly accurate," are based on questionnaire returns from neurologists throughout the country and apply to "clinic or private patients having convulsive seizures."²² They may not apply to institutional cases. Sixty per cent of 1,869 patients had only one type of convulsion, 40 per cent more than one, and 2.5 per cent all three. Fifty-one per cent had only grand mal, 8 per cent only petit mal, and 1 per cent only psychic seizures. Including those subject to more than one type, 90 per cent had grand mal, 45 per cent petit mal, and 8 per cent psychic attacks. Females were more susceptible to petit mal and males to grand mal.

Based on a study of 1,750 epileptics, the average annual number of grand mals was 114 as against 573 petit mals and 217 psychic seizures. Of 1,567 epileptics with grand mal, 25 per cent had less than five a year, 50 per cent less than 15, 75 per cent less than 50, 22 per cent more than 1,000, and 0.4 per cent more than 5,000, with the maximum for a one-year period of 16,000 and for a fourteen-year period, 125,000. Of 657 petit mals, about 25 per cent had less than 20 a year, 45 per cent less than 50, 18 per cent more than 1,000, and 1.2 per cent more than 5,000. The maximum frequencies were

²¹ Robinson, Leon J., and Osterheld, Roger G., "The Electroencephalogram in Epileptic Patients Aged Five to 80 Years," *Journal of Nervous and Mental Diseases*, 1917, 106:464-470.

"Kappa waves," allegedly connected with thinking, have been located in the temporal lobes. Kennedy, John L., Gottsdanker, Robert M., Armington, John C., and Gray, Florence E., "A New Electroencephalogram Associated with Thinking," *Science*, November 12, 1918, 527-529.

²² Lennox, *Science and Seizures*, pp. 34 ff.

25,000 for a four-year period and 210,000 for a 29-year period, the latter patient having also had about 400 grand mals. The ratio of patients with 1,500 petit mals a year was five times greater for children under five than for adults over twenty; whereas the corresponding figure for the grand mal type was three times greater for the children. Obviously attacks occur much more frequently among children than among adults.

Although the data from different investigators are somewhat discrepant, no marked sex difference exists in this disorder. The slight preponderance found among males can perhaps be ascribed to more birth injuries among male infants and more head trauma among male adults. Twelve and a half per cent of William Gower's cases in England developed recognizable symptoms before the age of three and about 75 per cent before twenty; 38.5 per cent of William Spratling's cases, also in England, developed before ten, 43.5 per cent from ten to twenty, and 82 per cent under twenty. The peak age for 7,350 inmates in the Craig Colony for Epileptics in New York state was below five years and from ten to fifteen years for 844 noninstitutional cases. The onset occurred before the age of twenty for 71 per cent of Lennox's cases.²³ Sixty-three per cent of the girls had the first convulsion before the age of fifteen, as against 52 per cent of the boys. The disorder is essentially one of childhood and youth with earlier onset among the girls.

The epileptic personality and temperament

Some of the earlier writings, based on observations of institutional cases, ascribed certain personality defects or abnormalities to epileptics, especially adults, either as a result of a constitutional diathesis or the disorganization or disintegration produced by the attacks. The reputed characteristics include emotional instability, impetuosity, shallowness of feeling or sentiment, monotony of speech and perseveration

²³ *Ibid.*, p. 38.

of stereotyped phrases, egotism, pedantry, fickleness, unreliability, moroseness, sulkiness, irritability, peevishness, stubbornness, quarrelsomeness, cruelty, vindictiveness, mendaciousness, sex aggressions, oscillations of humor, mood, and character (unprovoked swings from extreme piety to blasphemy), temper tantrums, violent outbreaks of irresponsibility, "epileptic furor" without loss of consciousness, and the like. Though such characterizations apply to a certain proportion of institutional cases (Spratling said from 75 per cent to 80 per cent) and a smaller ratio of noninstitutional epileptics, they are less the result of any innate temperamental peculiarity of the epileptic or of a specific inherited "epileptic personality" structure²⁴ in the idiopathic cases than of the ravages of uncontrolled seizures, especially of the grand mal type, or of the direct influence of cortical lesions, or of the toxic effects of certain anticonvulsant drugs, or of the social maltreatment to which the epileptic is often exposed—avoidance, scornful discrimination, exclusion from school, segregation in the home, and similar insults. Many nonepileptics possess some of the same characteristics, and the personality characteristics among epileptics are highly diverse. Moreover, successful cerebral operations, the administration of effective anticonvulsant drugs in nontoxic doses, and judicious social, psychological, and educational treatment have overcome many

²⁴ On this point, consult the summary of studies in Penfield, Wilder, and Erickson, Theodore C., *Epilepsy and Cerebral Localization*. Springfield, Ill.: Charles C. Thomas, 1941, pp. 560 ff.; this section was prepared by Mary R. Harrower-Erickson; pages 363-454 contain discussions, with illustrations and references, of roentgenography (X-rays of the brain), pneumoencephalography, and electroencephalography. See also the references in the recent article by Lewinski, Robert J., "The Psychometric Pattern. III. Epilepsy," *American Journal of Orthopsychiatry*, October, 1947, 714-722 (also gives the profiles of the tests in the Wechsler-Bellevue for 25 idiopathic, grand mal males).

No evidence that epileptics constitute a distinct type intellectually appeared from the first survey of the inmates of an institution for epileptics by means of standardized psychological tests: Wallin, J. E. W., *Experimental Studies of Mental Defectives*. Baltimore: Warwick & York, 1912, p. 108. The laboratory of clinical psychology established by the writer in 1911 in the New Jersey State Village for Epileptics at Skillman was, according to report, the first of the kind established in an institution for epileptics anywhere in the world.

personality distortions and character obliquities in many epileptics.²⁵ This suggests that the maladjustments were not ineradicably fixed in the innate personality structure of the epileptic. Freed of their convulsions extra-institutional epileptics are "as a group," in the opinion of Putnam,²⁶ "outstandingly even-tempered, uncomplaining, and devoted, and . . . among them are to be found many unusually stable, intelligent, and delightful personalities." According to Lennox the majority of epileptics who frequent hospital clinics and the private offices of physicians "are no more peculiar than the 'run' of the population."²⁷

The relation of epilepsy to intellectual ability and mental efficiency

Superior epileptics. Epilepsy is compatible with all levels of intelligence from genius to idiocy. Some of the world's great and near-great in many walks of life have, reputedly, suffered from some kind of convulsive seizures: Lord Byron (mild seizures); Algernon Charles Swinburne (jerkings when excited, temper tantrums, periods of dipsomania, falling during attacks); Edward Lear, writer of limericks (seizures of almost daily occurrence); Blaise Pascal, French philosopher and mathematician; Hector Berlioz, French composer; Gustave Flaubert, French novelist (swoonings, fallings, and nocturnal convulsions); Guy de Maupassant, French writer; Julius Caesar (headaches and seizures); Alexander the Great; Peter the Great of Russia (lifelong mild and short attacks); Alfred the Great of England; William III of England; Louis XIII of France; Charles V of Spain; Emperor Caligula of Rome; Mohammed, founder of the Moslem religion, who regarded his seizures as evidence of divine inspiration; an

²⁵ See, e.g., Deutsch, Leopold, and Wiener, Louise L., "Children with Epilepsy: Emotional Problems and Treatment," *American Journal of Orthopsychiatry*, January, 1948, 65-72.

²⁶ Putnam, Tracy J., *Convulsive Seizures, How to Deal with Them*. Philadelphia: J. B. Lippincott Company, 1943, p. 32.

²⁷ *Science and Seizures*, p. 58.

American psychologist and a Canadian philosopher, known to the author, reported to have been subject to some form of convulsive attacks.

Mentally impaired epileptics. On the other hand, many epileptics are definitely mentally defective, especially institutional cases, either as a result of the mental deterioration (dementia) produced by severe, frequent, long continued grand mals, or more frequently, as a complication superimposed upon a background of inherited mental defectiveness. In the latter case, epilepsy is not the cause of the mental defectiveness but it may produce progressive mental deterioration, adding dementia to amentia. The outlook for mental and educational improvement in such cases is less hopeful; epileptic spells apparently produce more mental impairment in the mentally deficient than in the normal. Epilepsy is a frequent complication of mental defectives of the lower grades (idiots, imbeciles, and microcephalics, exclusive of cretins and mongols)²⁸ and of those subject to lesions of the motor cortical area, as shown by post mortems or by electroencephalograms or by pneumoencephalograms (sclerotics, paralytics or paretics, syphilitics, hydrocephalics, and porencephalics). Spratling in 1904 estimated that 50 per cent of epileptics (probably institutional cases) become feeble-minded.²⁹ Tredgold gives the following figures in 1937 (based on an earlier investigation of "epilepsy of the simple variety," largely institutional cases): 70 per cent of patients "presenting signs of gross lesions"; 37 per cent of primary aments (idiopathic cases without gross cerebral lesions); 11 per cent of the "feeble-minded" (morons); 42 per cent of imbeciles; and 56 per cent of idiots.³⁰ According to Penrose, writing in 1934, about 20 per cent of institutional mental defec-

²⁸ The writer has never known of an epileptic mongol or cretin. None of Yarnet's seven cretins, and but two of his 92 mongols (2.2 per cent) were epileptics.

²⁹ For a summary of other older findings and estimates see Wallin, "The Diagnostic Findings"; and *Problems of Subnormality*. Yonkers: World Book Company, 1917. pp. 361 ff.

³⁰ Tredgold, *Textbook of Mental Deficiency* (6th edition), 1937, pp. 241 f.

tives are epileptics, most having convulsions throughout life.³¹ Yannet, in 1945, reported convulsions in only 6.6 per cent of his familial cases, in 18.4 per cent of "undifferentiated" inmates, in 32.2 per cent of the cerebrally palsied, in 35.7 per cent of the traumatic cases, and in 41.2 per cent of infection cases. Convulsions were found in 21 per cent of idiots, 16 per cent of imbeciles, and 9 per cent of morons—decidedly smaller percentages than those reported by Tredgold, possibly because of the more effective treatment recently discovered. Yannet attributes less than 4 per cent of the mental defect in idiopathic epileptics to recurrent convulsions.³²

Test results. In the pioneering study in 1910-11 of all the inmates, 333, of the New Jersey State Village for Epileptics, by means of the Binet tests (1908 version), before the advent of the I. Q., the writer classified two-thirds of the inmates as mental defectives, purely upon the basis of the tests. And almost as many of the brightest pupils in the school department were classified as mental defectives on the basis of the scores made in a battery of specially devised group intelligence tests also administered to public school children of various ages. Of the adults over twenty-one years of age (70 per cent of the examinees), 4 per cent had Binet ages less than three, 22 per cent had Binet ages from three to seven, 65.4 per cent from eight to twelve, and 6 per cent reached age thirteen. On the basis of more conservative standards of diagnosis adopted by the writer a year or two later, about 45 per cent of the adults would have been classified as borderline, backward, and normal, leaving about 55 per cent of this group to be classified as mental defectives.³³ About two-thirds of the epileptic children examined some years later in the St. Louis psychoeducational clinic (which was primarily con-

³¹ Penrose, *Mental Defect*, p. 138.

³² Yannet, "Diagnostic Classification," pp. 83 f.

³³ Wallin, "Experimental Studies of Mental Defectives," pp. 12 ff.; "The Measurement of Mental Traits in Normal and Epileptic School Children" (1923); *Problems of Subnormality*, pp. 350-381. These references contain an analysis of the comparative performance of epileptic and normal children in different kinds of psychological tests.

cerned with problems of mental defect and retardation) were classified as mental deficient.³⁴ These children, it must be borne in mind, were referred to the clinic primarily because of their mental limitations.

Later studies, based upon more conservative standards, the use of the I. Q., and, more recently, more effective medical therapy, have yielded lower ratios, especially for noninstitutional referrals.

In 1924 Fox reported a median Burt-Binet of 71 for 99 epileptic boys and 63 for 51 epileptic girls, ages five to sixteen, all admitted as "capable of some education and occupation" to the Lingfield Epileptic Colony in England.³⁵

Dawson and Conn found a mean Burt-Binet I. Q. of 80, with a range of from 49 to 117, for the 49 epileptic children, ages four to twelve, in the Royal Hospital for sick children in Glasgow.³⁶ Fetterman and Barnes found a mean Stanford-Binet I. Q. of 74 for 105 epileptic children and adults, mostly idiopathic cases, examined in a hospital dispensary, with a range of from 34 to 133, and 40 per cent below 70.³⁷ Sullivan and Gahagan reported a median I. Q. of 92, ranging from 11 to 141, for 103 epileptic children in the Children's Hospital in Los Angeles who were given the Stanford-Binet, Kuhlmann-Binet, and Gesell Normative Schedules of Development.³⁸ Somerfield-Ziskind and Ziskind found a mean I. Q. of 93 for 100 out-patient cases in Los Angeles, ages three to fifty-eight, given a battery of psychological tests, including the Stanford-Binet.³⁹ The mean was 97 for the idiopathic and 88

³⁴ Wallin, "The Diagnostic Findings," p. 185.

³⁵ Fox, J. Taylor, "The Response of Epileptic Children to Mental and Educational Tests," *British Journal of Medical Psychology*, August, 1924, 235-248.

³⁶ Dawson, Shepherd, and Conn, J. C. M., "Intelligence of Epileptic Children," *Archives of Diseases of Childhood*, 1929, 4:142-151.

³⁷ Fetterman, Joseph, and Barnes, Margaret R., "Serial Studies of the Intelligence of Patients with Epilepsy," *Archives of Neurology and Psychiatry*, 1934, 797-801.

³⁸ Sullivan, Ellen B., and Gahagan, Lawrence, "On the Intelligence of Epileptic Children," *Genetic Psychology Monographs*, 1935, 5:309-376.

³⁹ Somerfield-Ziskind, Esther, and Ziskind, Eugene, "Effect of Phenobarbital on the Mentality of Epileptic Patients," *Archives of Neurology and Psychiatry*, 1940, 43:70-79.

for the symptomatic cases. The Stanford-Binet I. Q. range for 66 idiopathic epileptics in the Dixon, Illinois, State Hospital, ages eight to fifty-three (80 per cent over twenty-four years) varied from the I. Q. interval 30 to 39 to 120 to 129, with a mean of 75. The mean I. Q. of those with major attacks was 65.8 and for those with minor attacks 75. Thirty-six and five-tenths per cent of the Dixon cases were classified as feeble-minded "in contrast to 22.4 in Dawson and Conn's group and 18.4 in Sullivan and Gahagan's group," and 1.5 per cent as superior in contrast with 6.1 per cent for Dawson and Conn's and 19.3 per cent for Sullivan and Gahagan's group.⁴⁰ Baker reported an I. Q. range (test used not stated) during a three-year period of from 70 to 90 for the "greatest number" of children in the Detroit Special Public School for Epileptics.⁴¹

On the basis of tests of 186 co-twins by means of Form L of the Stanford-Binet or the Wechsler-Bellevue, Lennox and Collins found a mean I. Q. of 108 for 149 nonepileptic twins, 96 for 27 epileptics without evidence of brain lesions, and 77 for 10 epileptics with brain injury.⁴² (The results are not given separately for the two scales of tests.)

Though repeated severe seizures, especially of the grand mal type, may produce mental deterioration, even mental defectiveness, in the mentally normal, it is difficult to formulate any generalization applicable to all cases. The rate of deterioration varies enormously with individuals. Some do not deteriorate at all, but improve mentally. Some authorities believe that the majority of the whole group of the epi-

⁴⁰ Hilkevitch, Rhea R., "A Study of the Intelligence of Institutionalized Epileptics of the Idiopathic Type," *American Journal of Orthopsychiatry*, April, 1946, 262-270.

⁴¹ Baker, Harry J., *Introduction to Exceptional Children*. New York: The Macmillan Company, 1944, p. 306.

⁴² Lennox, W. G., and Collins, A. Louise, "Intelligence of Normal and Epileptic Twins," *American Journal of Psychiatry*, May, 1945, 764-769.

For reference to other investigations see Pintner, Rudolf, Eisenson, Jon, and Stanton, Mildred, *The Psychology of the Physically Handicapped*. New York: F. S. Crofts & Co., 1941, pp. 297-316; Penfield and Erickson, *Epilepsy and Cerebral Localization*, pp. 548-552.

leptic is mentally normal and that the majority of at least the idiopathic cases will remain such in spite of their nervous attacks.

Dawson and Conn reported a mean loss of 15.5 I. Q.'s (from 82 to 66.5) for 21 children between the first and second testing at intervals of from eight months to five years. Fetterman and Barnes found that 19 of 46 retests showed a "slight increase," 23 a "moderate loss," and 4 no change. Thirty-seven per cent of 130 of Fox's cases retested with the Burt-Binet after a year had a lower score, 22 per cent a higher score, 41 per cent approximately the same score, and only 8 per cent showed marked deterioration.

A second test of 26 of the Dixon epileptics after intervals of one to eleven years showed a mean drop of 4.2 I. Q.'s, with extreme variations of from minus 44 to plus 24. Nine displayed an average loss of 19 points, and 17 had remained stationary or had improved. The mean I. Q. of those with infrequent or moderately frequent attacks was 77 and for those with "extremely frequent" attacks 60. Paskind reached the conclusion, based on the ability to continue in their positions, that only 6 per cent of 304 private, extra-institutional epileptics who had suffered attacks for six years or more (15.4 per cent over 25 years) had deteriorated. Ninety-three and five-tenths per cent were reported to be in "excellent mental and physical health." For 112 cases the condition at the time of the report was determined by questionnaire returns. No psychological tests were administered.⁴³

Lennox states that 67 per cent of 1,640 clinic and private (noninstitutional) idiopathic cases were mentally normal, 23 per cent slightly subnormal, 9 per cent "definitely deteriorated," and only 1 per cent "markedly deteriorated."⁴⁴ This appraisal of the intelligence level is, apparently, based on estimated intelligence rather than tested intelligence. Seventy-

⁴³ Paskind, Harry A., "Extramural Patients with Epilepsy with Special Reference to Frequent Absence of Deterioration," *Archives of Neurology and Psychiatry*, August, 1932, 370-385.

⁴⁴ Lennox, *Science and Seizures*, p. 51.

five per cent of 1,638 patients normal at birth "were normal when seen by the examining physicians," whereas only 7 per cent were "definitely deteriorated." Among patients who "seemed mentally normal at birth," who had had over 1,000 convulsions, 40 per cent of grand mal cases and 83 per cent of petit mal cases remained mentally normal.⁴⁵ Nine persons with over 10,000 grand mals each remained mentally normal. Nevertheless, though 75 per cent of 1,899 clinic cases of all types were mentally normal at the onset of the attacks, only 46 per cent of those subject to seizures for twenty-five years or more were normal. Only 54 per cent of those with brain injuries prior to the onset of the convulsions, however, were normal compared with 67 per cent of the essential type. The psychic seizures produce as much mental impairment as the grand mal. These conclusions apparently are based on estimated rather than on measured intelligence.⁴⁶

⁴⁵ *Ibid.*, pp. 53 f.

⁴⁶ Spratling's statement many years ago that the major attacks do not impair mentality as much as the lesser ones is not borne out by later investigations.

Chapter 15

THE CAUSATION AND TREATMENT OF EPILEPSY

Causation of idiopathic epilepsy

The older estimates of the degree to which epilepsy was hereditary varied from 2 per cent (Reed) to 87 per cent (Kraepelin),¹ depending on whether the individual's concept of heredity included "polymorphism" or dissimilar heredity—the inheritance of a unit factor predisposing to the development of almost any neuropathic disorder in the family lines—or was restricted to similar heredity, that is, epilepsy in the relationship. Spratling's statement that "similar heredity is the chief cause of epilepsy," has not been substantiated. Reed found only 2 per cent of similar heredity, Spratling himself only 16 per cent, and Kraepelin only 22 per cent (mother or father epileptic). More recently Stein reported convulsions in 18.1 per cent of the immediate families of 1,000 epileptics in the Massachusetts State colony as against only 4.6 per cent in a group of 722 controls.² Paskind found epilepsy in only 7.5 per cent of the family histories in his 304 extramural cases, although he classified 60 per cent as hereditary, based on the finding of dissimilar neuropathic traits in the pedigrees.³

In an investigation of 253 pairs of twins, Conrad found that

¹ For a summary of the older estimates see Wallin, *Problems of Subnormality*, pp. 355 f. and Paskind, "Extramural Patients," pp. 378 f.

² Stein, Calvert, "Hereditary Factors in Epilepsy: Comparative Study of 1,000 Institutionalized Epileptics and 1,115 Non-Epileptic Controls," *American Journal of Psychiatry*, March, 1933, 12:989-1037.

³ Paskind, *op. cit.*, p. 381. Consult Paskind for additional statistics on this point.

epilepsy occurred in both identical (uniovular) twins in 86.3 per cent of the idiopathic cases. The corresponding figure for the fraternal twins was 3 per cent.⁴ Identical or similar twins, which are always of the same sex, have the same heredity because they are the product of a single fertilized ovum. Since all the cells from the same fertilized ovum have the same gene constitution and since the twins are very similar in mental and physical traits, any differences that develop in such twins must be due to differences in environmental impacts or in maturation. Fraternal or nonidentical twins, often of dissimilar sex and appearance, are derived from two separate ova fertilized at the same time by two different spermatozoa. They are genetically no more alike than any siblings born at different times, although they have the same prenatal environment. About three births in a thousand are identical twins, and about nine in a thousand births are fraternal twins. The study of the physical, mental, and social traits of identical twins reared in superior and inferior cultural and physical environments affords, it is believed, a crucial test of the relative influence of heredity and environment, and many such investigations have been made.

The most compelling evidence in favor of the hereditary hypothesis is derived from the recent electroencephalographic studies of the cortical activity of epileptics. The limited space here available permits only a succinct summary of the conclusions of one of the chief contributors in this field.⁵

⁴Quoted from Penfield and Erickson, *Epilepsy and Cerebral Localization*, pp. 309 f. Consult their Chapter XI on the inheritance of epilepsy.

For references to similar investigations and pedigree studies see Landis, C., and Bolles, M. M., *Textbook of Abnormal Psychology*, pp. 264, 274 f. For a critical review of some hereditary studies see Myerson, et. al., *Eugenical Sterilization*, pp. 144-244.

⁵Lennox, *Science and Seizures*, especially pp. 98 f.; "The Genetics of Epilepsy," *American Journal of Psychiatry*, January, 1947, 457-462. Two of the pioneering investigations of the encephalograms of the relatives of epileptics were made by Löwenbach, Hans, "The Encephalogram in Healthy Relatives of Epileptics," *Bulletin of the Johns Hopkins Hospital*, July, 1939, 125-137; and by Strauss, Hans, Rahm, W. E., Jr., and Barrera, S. Eugene, "Electroencephalographic Studies in Relatives of Epileptics," *Proceedings of the Society for Experimental Biology and Medicine*, 1939, 42:207-212.

Based on the cortical electric wave studies of the relatives of a limited number of epileptics, on the family histories of almost 2,000 extra-institutional epileptics treated by neurologists in different parts of the country, on 425 cases of migraine⁶ treated at the Boston City Hospital, on 1,000 controls from the general population (medical students, nurses, and miscellaneous patients), and on the incidence of epilepsy in the 1917 military draft (one in 200 were epileptic rejects),⁷ Lennox has reached the conclusion that 0.5 per cent of the general population is subject to epileptic seizures and 5 per cent suffer from migraine. This would yield from 500,000 to 700,000 epileptics throughout the country. This estimate contrasts sharply with the earlier estimates of from about 1 to 5 per 2,000 of the general population.⁸ The incidence of epilepsy was 2.8 per cent among 12,119 parents, siblings, and children of the 2,000 epileptics, or 5.5 times greater than in the general population. The incidence was 60 per cent less among the relatives of those who had symptomatic epilepsy.

Dysrhythmia was present in nearly all epileptics. Moreover, the electrical brain wave records were found abnormal for over 90 per cent of the one or the other parent of 55 unselected epileptics. In 30 per cent the brain waves were abnormal for both parents. Sixty per cent of 183 near relatives of 94 epileptics showed abnormal brain waves. One in about 36 near relatives of the "average" epileptic had seizures, and one in 12 had migraine.

⁶ Migraine, popularly known as sick headache or "bilious headache," a recurrent type of headache ordinarily located on one side or in one area of the head, very painful, and accompanied by upset stomach, is interpreted as genetically related to epilepsy—"an epilepsy of the vegetative nervous system." Some suffer from both kinds of seizures; some change from one to the other. The number of migraine sufferers in the United States is estimated at six million. It is affirmed that more epileptics come from the migraine population than from the epileptic population.

⁷ The same ratio of epileptic rejects obtained among over nine million selectees from eighteen to forty-four years of age, physically examined by selective service boards between April, 1942 and December, 1943.

⁸ Wallin, "The Diagnostic Findings," p. 185.

In 78 per cent of 2,129 cases of essential epilepsy, no cause could be found other than an inherited predisposition toward dysrhythmia or the electro-physico-chemical condition responsible for the low spasmophilic threshold. Of the remaining 22 per cent of "symptomatic" cases, 5.6 per cent had a congenital or birth-injury brain defect; 5.7 per cent had a postnatal brain lesion; 4.2 per cent had been afflicted with a brain infection; 2.6 per cent had a tumor; 1.9 per cent suffered from faulty circulation in the brain; 1.5 per cent revealed various other brain lesions; and influences outside of the brain were held accountable for only 0.9 per cent of the cases. It is conceded that some of the acquired causes played merely a role contributory to the underlying constitutional predisposition.

The broadest generalization suggested by these data is that about 10 per cent of the general population are subject to brain wave disturbances similar to those found in epileptics (more than ten million persons in the United States), that only 5 per cent of those predisposed actually become epileptic, that about 75 per cent of those who do develop epilepsy are constitutional cases, and that seizures can be prevented in many of these cases, as well as in many of the symptomatic cases. The assumption is that potential epileptics can be protected against the ravages of the exciting or contributory causes of the disorder. These sweeping conclusions require confirmation from brain wave and follow-up studies on a larger scale.

The medical treatment of the epilepsies

Almost innumerable remedies, nostrums, gruesome concoctions, severe exorcisms, and superstitious practices have been prescribed from time to time for the alleviation or cure of this puzzling malady. The modern anticonvulsants have superseded almost all of the earlier forms of treatment, even those found valuable in certain cases, such as dehydration

(restricted water intake), starvation, the ketogenic dietary,⁹ or the bromides, first used in 1857 (which often produce bromism: skin rashes, drowsiness, and depression). The best known among the newer drugs include: luminal (gardenal), the trade name for the sedative drug phenobarbital, effective against grand mal (first used by Alfred Hauptmann in Germany in 1912); and dilantin, the trade name for the non-sedative phenytoin sodium, effective against grand mal and also against psychomotor seizures (first used by Tracy Putnam and Houston Merritt in Boston on human patients in 1938). Another nonsedative drug, tridione, has proved effective against pure petit mal cases (first used by Lennox on human patients in 1945),¹⁰ but it is a dangerous drug. It sometimes produces mild to severe secondary anemia and requires routine blood counts. Given in proper dosage with utmost regularity, these synthetic drugs eliminate about 75 per cent of the convulsions in about 75 per cent of the cases. In proper dosage none of these drugs produces any mental deterioration or any other permanent injury. To cite the results of one investigation: based upon the repetition of the same battery of psychological tests after a year's interval, the Ziskinds found no significant mean I. Q. change in 42 untreated cases and 48 cases treated with phenobarbital (luminol), except for children under age sixteen. Those treated in the younger group gained sixteen months in test age as compared with twelve months for the untreated group. Doses of 1½ grains

⁹ A diet rich in fats and poor in proteins (meats) and carbohydrates (starches and sugar) and restricted water intake. Such a diet (as well as fasting) produces ketone acids in the blood, which exert a soothing influence on the nerves and reduce the grand mal and petit mal seizures in from 33 per cent to 50 per cent of idiopathic cases. It is particularly potent against petit mal, especially with children. See Talbot, Fritz B., *Treatment of Epilepsy*. New York: The Macmillan Company, 1930; Peterman, "Convulsions in Childhood," pp. 408 f.

¹⁰ A reduction of convulsions and mental improvement has also been reported from the use of glutamic acid in the case of petit mal and psychomotor attacks: Price, Jerry C., Waelsch, Heinrich, and Putnam, Tracy J., "dl-Glutamic Acid in the Treatment of Petit Mal and Psychomotor Seizures," *Journal of the American Medical Association*, November 24, 1943, 1153-1156.

of phenobarbital two or three times daily have not produced any mental deterioration within a two-year period.¹¹ Temporary annoyances can usually be overcome by change of dosage or by replacement with a succedaneum (a drug that may be substituted for another of similar properties). Some respond better to dilantin than to luminal (which is not effective against the psychomotor attacks); with others it is *vice versa*. Although the effects of the drug treatment are in most cases merely palliative, permanent cures sometimes result when the drug normalizes the brain waves. A case may be regarded as cured if the attacks do not recur after the drug has been withdrawn—ordinarily after several years of treatment.

A limited number of carefully selected epileptics can be relieved of their seizures and perhaps have their mental condition restored to normality by the surgical removal of focal tumors, scars, adhesions, or localized bone pressures. Brain operations are now no more dangerous at the hands of the skilled neurosurgeon than are abdominal operations. They can be performed with the use of a local anesthetic. Operations on the brain and skull are painless. Nevertheless, the possible dangers involved in brain surgery include postoperative convulsions, paralysis, and possibly death. Some of the war-wounded soldiers respond favorably to anticonvulsants (phenobarbital and dilantin).¹²

A grand mal convulsion requires no attention at the time of the paroxysm other than assisting the patient to break the fall; inserting a folded handkerchief between the molars to prevent tongue biting; if the facial muscles contort, loosening the clothing (collar, neckband, belt); and allowing the patient to lie on his back on the floor undisturbed, with the head turned to one side to allow drainage of the saliva.

¹¹ Somerfield-Ziskind and Ziskind, "Effect of Phenobarbital," p. 78.

¹² Walker, A. Earl, and Quadfeschel, Fred A., "Follow-Up Report on a Series of Posttraumatic Epileptics," *American Journal of Psychiatry*, June, 1948, 781-782.

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"The Petit Mal Epilepsies, Their Treatment with Tridione," *Journal of the American Medical Association*, December 15, 1945, 1069-1073; *ibid.*, May 10, 1947, 138-143.

Another drug (mesatoin) has been found effective with some cases.

Psychological and social treatment

In 1910 the writer requested about a dozen epileptologists, neurologists, and psychiatrists to offer suggestions regarding the important problems that the new laboratory of clinical psychology in the New Jersey State Village for Epileptics ought to investigate. One psychoanalyst replied that the only psychological studies of epileptics of fundamental importance were psychoanalytic in character. Although psychoanalytic procedures have not been widely applied in this area since then, nevertheless, the psychological and social factors cannot be disregarded in the diagnosis and therapy of epilepsy. The relation of the psycho-social factors to the malady is twofold. First, emotional disorders and mental conflicts, even of a repressed nature, may serve as precipitants of seizures, particularly of the psychomotor types. Second, the morbid sensitivity about being afflicted with a mysterious and repulsive malady may be more inimical to the subject's

welfare and may constitute a greater bar to his happy social adjustment than the handicap of the physical attacks.

Most of the psychological difficulties, adjustment problems, and maladjustments exhibited by many epileptic children and adults are probably produced less by the ravages of the disease itself or by the putative "epileptic personality" than by the derogatory attitudes of parents, siblings, playmates, neighbors, and others, and the ill-advised and misdirected treatment or maltreatment to which many epileptics may be subjected by their associates and caretakers because of lack of understanding or prejudice. On the other hand, due to excessive misdirected or maudlin sympathy, the epileptic child may be excessively humored or coddled, or be over-protected by the parent (especially the mother). He may be allowed to have his own way in everything and everything may be done for him, and he may be given no responsibilities in assisting in the care of the home or in taking care of himself. In consequence, he may become egocentric and selfish and may fail to develop initiative, resourcefulness, self-helpfulness, and independence, and thus remain emotionally and intellectually immature. At times he may become restive and resentful because of the excessive "mothering" and become aggressive or disorderly in order to acquire status and liberty of action. On the other hand, his psychomotor attacks may be interpreted as wilfulness, contrariness, or malicious mischievousness and he may be subjected to harsh criticism or severe punishment. Because of the parents' ignorance, misconception of the nature of the nervous attacks, or mistaken family pride, the child may be hidden away in some room or kept at home so that the neighbors will not find out anything about the character of his affliction. He may thus be deprived of all opportunity to lead a normal social life in association with other children, even his own siblings. As a result of social ostracism he may become seclusive, timid, unsocial or antisocial. He may become embittered and resentful because of the favoritism shown his brothers and sis-

ters and the discriminating practices to which he is subjected. He may be teased, snubbed, reviled, or shunned by siblings and companions and may be driven to compensate for his frustrations by becoming aggressive and rebellious, and by indulging in temper tantrums; or he may become a victim of self-pity, and grow despondent and develop deep-seated feelings of inadequacy and inferiority. Thus he may voluntarily withdraw from public gatherings and social contacts to avoid the embarrassment produced by his seizures. Because of the parents' attitude of hopelessness regarding the prognosis of the malady, he may be neglected and be denied the advantages of proper medical and psychological treatment and suffer deterioration because of lack of stimulation. He may be excluded from the advantages of public school education just because he is an epileptic or because of the confusion and excitement produced by possible attacks in a classroom in which insight into his condition is usually lacking. Thus excluded he ordinarily receives very little if any formal instruction unless he is provided with a teacher at home or placed in a colony school. Because of the inability of the parents to develop an unemotional, realistic, and objective attitude toward the affliction, the child may become a victim of the irrational, emotionalized behavior of the members of the family precipitated by the sight of the seizures. He may become a hapless victim of the feelings of humiliation, anxiety, apprehension, fearsomeness, brooding, or insecurity manifested by the parents or by the other siblings. The feelings of distress, discouragement, hopelessness, or dread reflected in behavior patterns on the part of parents, siblings, and others are not unnoticed by the child. Severe emotional conflicts and feelings of frustration often develop in the adolescent because of vocational vexations: the difficulty of suitable job placement, job insecurity, financial worries, and problems of marriage. Although the physical, social, and economic plight of the epileptic has been very

grievous throughout the centuries, modern science has brought a new vision and a new hope for the least understood and the most neglected, perhaps, of all kinds of handicapped children.

What are some of the suggested remedies from the psychological vantage point? The paramount need calls for scientific enlightenment and realism in the attack on the problem of juvenile seizures. Parents, teachers, and the public in general should realize that epilepsy, although the underlying mechanism is not fully understood, is a disease of the nervous system of naturalistic origin, and is no more mystical, supernatural, repulsive, or disgraceful than an attack of hayfever, asthma, or migraine. It is no more fatal, in fact even less fatal, than many other bodily disorders, for instance, rheumatic heart disease, nephritis, or cancer. It does not produce more feeble-mindedness or insanity than do some other diseases of the brain, for example, encephalitis or meningitis. Most of the mentally defective epileptics are, based on test results, primary aments. Moreover, seizures tend to diminish spontaneously with advancing age, and most cases are susceptible to decided alleviation; some can be cured by recognized modes of medical or surgical treatment. As is the case with several other nervous diseases, proper medical, psychological, and educational treatment also brings about socially acceptable conduct in the majority of epileptic children. In the majority of cases epilepsy is not a hopeless malady. The worst cases are no more hopeless than the worst cases of encephalitis. Imbue parents and siblings with the conviction that there is no more stigma attached to epilepsy than to an attack of heart disease or biliousness, get them to adopt a calm, casual, matter of fact attitude toward seizures and their control, and get them to encourage the child and to fortify his morale. Adopt an attitude of frankness and sincerity in all dealings with the child. Tell him the truth about his disorder so far as the facts are known by

modern science and so far as is compatible with his level of comprehension, so that he may have a correct understanding of his condition.

The majority of epileptic children (some would say 75 per cent) are mentally normal and should be treated like normal children with a particular affliction. They crave the same affection, ego satisfaction, recognition of individual merit and personal rights, and the same security in home and school as any other children.

A part of the psychological treatment may involve a change of scene from a convulsion-provoking home, torn by discord and bickering, to a quiet, nonirritating institution. Restful, congenial, harmonious, well regulated living conditions conduce to nervous stability and serenity and are essential for the rehabilitation of nervously unstable children.

To prevent the deteriorating effects of idleness, introvert rumination, daydreaming, and indulgence in self-pity and glorification, and to relieve nervous tension, it is necessary to provide an abundance of vital challenges adapted to individual requirements and the objectification of the child's interest and attention, such as entertainments, varied recreational activities, club activities, outdoor and indoor games, sports, hikes, camping, hobbies, creative activities, occupational therapy, gardening, and farm activities. One of the unique advantages of the farm colonies is the opportunity they afford for all kinds of healthy outdoor work suitable to the interests and capacities of the young and the old of both sexes. Vigorous outdoor exercise (with proper controls for those with brain injuries and disease of the cerebral blood vessels) will improve the physical stamina of the child and serve as a healthy outlet for emotional tensions and pent-up energy.

A certain proportion of epileptics require institutional or colony care: some because they are psychotic; some because they are feeble-minded; some because of the continued severity of the attacks, which have not yielded to treatment; and some because they cannot receive in the home the care, pro-

tection, treatment, and opportunities they require. In 1945, 26,040 epileptics were in state and private (679 cases) institutions and colonies for epileptics and for the feeble-minded throughout the country. Similar data are not revealed in the statistics from hospitals for psychotics, but of the new admissions that year to all such institutions, 1,675 were psychotics with convulsive disorder and 656 were epileptics without psychosis.

All epileptics should be given thorough physical examinations, including electroencephalographic studies, early in the course of the disease, with repeat examinations as indicated. Pneumoencephalograms should be obtained whenever there is reason to suspect gross cerebral injuries. Psychological examinations should be given routinely. Diagnosis and therapy by means of interview and play techniques and psychiatric and psychoanalytic procedures should be afforded as required. Many epileptics need psychological assistance in the solution of their emotional and personality problems.

The technique of modern brain photography should also be applied to persistently unruly, ungovernable, and intractable children. Episodic temper tantrums or behavior disorders may be of the nature of epileptic equivalents or may be provoked by discoverable brain lesions. Abnormal brain waves were found in 71 per cent of 71 behavior problem children of the more intractable kind domiciled in the Bradley Home for Children in Providence, Rhode Island. The electrical abnormalities were marked in 59 per cent. In 39 per cent the waves were "epileptoid" in character although no child at the time was subject to seizures and only two had previously betrayed any suspicion of epilepsy.¹³ The children in the latter group betrayed "characteristic mental and emotional disturbances analogous to those often associated with the epileptic personality." Nine children diagnosed

¹³ Jasper, Herbert H., Solomon, Philip, and Bradley, Charles, "Electroencephalographic Analyses of Behavior Problem Children," *American Journal of Psychiatry*, 1938, 95:641-658; Knott, John R., *Psychological Bulletin*, 1941, 944-975.

as "schizoid" (withdrawn, seclusive, introvertive, unsocial) displayed definitely abnormal rhythms although not epileptoid. Slow dysrhythmias (abnormal waves) predominated, such as go with decreased blood sugar, or carbon dioxide, or structural alterations in the cortical neurones. During a follow-up period of from one to four years only the children with "normal brain potentials," made a complete behavioral adjustment. Most of those with abnormal waves made no improvement or only fair improvement, some as a result of psychiatric treatment and some as a result of the use of the drug benzedrine.¹⁴

Two years later clinical and electroencephalographic studies of 44 children, ages 3.5 to 13, referred to the Children's Service of the New York State Psychiatric Institute as psychiatric cases, revealed abnormal encephalograms in 68 per cent of the cases. The dysrhythmia was of the diffuse type in 20 (45 per cent) and focal in 7 (16 per cent) of the cases. The pneumoencephalograms proved to be abnormal in only four of the six focal cases studied by this technique, indicating that the electroencephalogram disclosed more brain injuries, as confirmed by clinical findings, than the pneumoencephalogram. Most of the cases were clinically classified as "primary behavior disorder" (22 cases), followed by organic brain disease (5 cases) and epilepsy (4 cases). However, the major grouping by one of the investigators was into "epileptoid personalities," 35 cases, of whom 25 showed abnormal brain waves similar to those found in epileptics. "Some epileptoids are biologically related to epileptics although the exact nature of the relationship is as yet unknown." ¹⁵ This

¹⁴ Bradley, Charles, "The Behavior of Children Receiving Benzedrine," *American Journal of Psychiatry*, 1937, 577-585. See also Walker, Charlotte, and Kirkpatrick, Barbara, "Dilantin Treatment for Behavior Problem Children with Abnormal Electroencephalograms," *American Journal of Psychiatry*, January, 1947, 487-492.

¹⁵ Strauss, H., Rahm, W. E., and Barrera, S. E., "Studies on a Group of Children with Psychiatric Disorders: I. Electroencephalographic Studies," *Psychosomatic Medicine*, January, 1940, 34-42.

conclusion would seem to confirm the old assumption that epileptics possess a peculiar personality constitution.

Some cases of incorrigibility and some minor delinquencies are doubtless due to pathological brain states or abnormal chemical functioning of the cerebral mechanism. The encephalographic techniques (electro- and pneumoencephalographic) should make it possible to discover at least some of these cases and to provide more intelligent medical, psychological, and educational treatment for them. Such cases call for skilled application of remedies rather than the employment of negative, repressive, or penal measures.

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Education and training

No child with seizures should be denied the privileges of public school education merely because he is an epileptic. No epileptic should be excluded from school unless his grand mal convulsions (and occasionally psychomotor attacks) occur in the daytime and remain uncontrolled and unless he is too low mentally to profit from public school attendance.

Before a child with occasional grand mal attacks is excluded he should be allowed a period on probation in a class for the mentally retarded. In the tranquil atmosphere of a well ordered special class taught by a sympathetic, understanding teacher-social worker some epileptics will experience remissions of their attacks. Moreover, under the stabilizing example of a calm, resourceful teacher the other pupils soon become adjusted to an occasional spell. In fact, they can readily become so conditioned by the teacher's attitude and behavior that they will become genuinely interested in the child's plight and willing to assist in any required succor. To minister to a child in distress is one of the most effective means of socialization and developing the spirit of cooperation among both young and old. Nearly all children, and adults as well, can be so conditioned that they can witness convulsions without becoming emotionally upset.

The mentally normal group should be given every advantage consonant with their individual needs in the regular grades, including a high school and college education for those possessing the needed interest and aptitude. The subnormals should be admitted to special classes for the mentally retarded suited to their intelligence level. In large cities a public day school for epileptic children might well be maintained on the pattern of the White Special School established by the Board of Education in Detroit in January, 1935 for children with diurnal seizures. This school was conducted as a day and residential (hospital) school until June, 1939, and since that time has continued as a day school only. Seizures in the "quiet régime of the epileptic school," where the attacks are taken as a matter of course by the teachers and pupils, are about 50 per cent less frequent than they were before. Most of the children are returned to the regular grades free of seizures after an average of one year in the special school.¹⁶

¹⁶ Baker, Harry J., *Introduction to Exceptional Children*. New York: The Macmillan Company, 1944, p. 315.

Although the advantages of systems of liberal arts and professional education should be freely afforded intellectually superior and talented epileptics on the same terms as other youths of equal ability, in many cases the emphasis must be on eminently practical forms of motor education and training, geared to the youth's interests and vocational prospects. The prejudice against the employment of epileptics has abated somewhat because of the demonstrated proficiency of epileptics whose seizures have been brought under control in many kinds of jobs in which an occasional seizure does not constitute a grave hazard, as would be the case in operating elevators, automobiles, trains, or airplanes, or working at heights, or with unprotected moving machinery, or near hot objects or bodies of water.

Most epileptics can be successfully adjusted to a wide range of jobs suited to their interests and possibilities. A follow-up investigation of 1,000 adult epileptics showed that about 75 per cent were successfully employed as farmers, laborers, mechanics, factory workers, plumbers, interior decorators, dressmakers, clerks, stenographers, beauticians, actresses, salesmen, dancers, writers, doctors, engineers, and teachers.¹⁷ Bement F. Hibbard reported in 1945 that 80 per cent of "ambulatory epileptics of employable age and under modern medical treatment" were successfully employed as accountants, typists, bookkeepers, laboratory technicians, shipping clerks, farm laborers, and skilled and unskilled laborers. Very few industrial accidents were reported. An additional 5 per cent with frequent or severe convulsions were employable under sheltered work-shop conditions.¹⁸

It is fortunate that a wide range of jobs is available almost everywhere—in industry, agriculture, horticulture, floriculture, animal husbandry, and home service—suitable to the interests and possibilities of epileptics and in which, under

¹⁷ Lennox, William G., and Cobb, Stanley, "The Employment of Epileptics," *Industrial Medicine*, December, 1942, 571 f.

¹⁸ *Are Epileptics Employable?* National Association to Control Epilepsy, undated bulletin.

skilled selective placement, they are able to make good. The most capable among them can occupy positions of responsibility in some of the higher professions. As with all persons, vocational pursuits adjusted to individual requirements possess definite therapeutic value, even as anticonvulsants in the case of the epileptic.

The outlook for epileptics has been decidedly advanced through their acceptance for rehabilitation services under the Federal Vocational Rehabilitation Act (Public Law 113, effective July 6, 1943), through the development of a comprehensive, coordinated plan of research, diagnosis, and medical and occupational treatment under the Veterans Administration, and through the organization of associations and agencies wholly dedicated to the investigation, care, and treatment of the epileptic, and the education of the public, such as the national laymen's organizations—The American Epilepsy League, Inc., in Boston, founded in 1939, and the National Association to Control Epilepsy in New York City, founded in 1944. The latter association, which for some years sponsored an epilepsy clinic in New York City devoted to the psychological, educational, vocational, and medical study and treatment of epileptic children, maintains an extensive nation-wide information and consultation service. The International League Against Epilepsy, with branches in many countries including the United States, was reorganized in 1935. Many states have organized very active state-wide programs in behalf of children and adults subject to convulsive seizures, often as a part of the activities of the state societies for the crippled. Among such states are California, Colorado, Indiana, Massachusetts (in cooperation with the Epilepsy Unit of the Boston Children's Hospital), Michigan, Ohio, Tennessee, Virginia (which maintains a state-wide clinic), and Washington. In many sections of the country epileptics have found their "place in the sun" of our social structure; in some sections they still reside in "darkest Africa," so far as social acceptance and modern educational and

medical treatment are concerned. The plight of the epileptic is still a major challenge to an enlightened citizenry!

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Chapter 16

CHILDREN AFFLICTED WITH ENCEPHALITIS¹

History

Encephalitis distinct from sleeping sickness. Few if any nervous maladies can produce greater permanent mental and physical devastation in children than the brain inflammation known variously as encephalitis (*encephalon*, brain; *itis*, inflammation), primary encephalitis, epidemic encephalitis, encephalitis lethargica, or sleeping sickness. The popular designation, "sleeping sickness," is a misnomer and should not be applied to this nervous disease. This term is properly applied to an endemic disease in equatorial Africa caused by the bite of the tsetse fly, and is characterized in the early stage by fever, headaches, and vomiting, and in the chronic stage by sleeplessness, lethargy, and eventually permanent coma and death. Many cases of prolonged sleeping or coma have been reported in the American public press which are different from the African form of sleeping sickness and which probably represent some form of encephalitis affecting the hypothalamus, a putative sleep center. The following cases supply illustrations of prolonged somnolence of this kind.

Patricia Maguire, often referred to as the "sleeping beauty" of Oak Park, Illinois, remained in a stupor or coma from February, 1932 (age 26) to September 29, 1937 (age 32), a period of five years and seven months. Maxine Yarrington, of Fairview, Pennsylvania, was asleep from September 24,

¹ This chapter has been read by Robert W. Graves, M.D., former Associate Professor of Neurology, Duke University, to whom acknowledgments are made.

1935 (age one year eight months) until she died in September, 1941, at the age of seven years eight months. She remained unconscious in a sort of coma six years. According to press reports, she sometimes babbled incoherently, but her wide-open eyes remained expressionless and unseeing. She is reported to have "awakened at times without knowing it." Dorothy June Russell, Wadsworth, Ohio, at the age of ten months was stricken with an attack of whooping cough lasting about six weeks, which was followed by convulsions and pneumonia. Recovery from the acute attack left her in a comatose condition in which she remained for six and a half years (she died in November, 1940 from a second attack of pneumonia) without regaining consciousness except that she seemed sometimes to be able to hear music on the radio and to be able to see and laugh. The most obvious post mortem findings were reduction of the brain weight and atrophy of the brain to about half size. The atrophy was particularly noticeable in the occipital cortex, which was very thin. The degenerated neurones in many areas are replaced by neuroglia cells.²

The term encephalitis lethargica was first applied by Constantin von Economo in 1917 to a disease of unknown origin (the pathogenic agent has not yet been definitely identified) which appeared in a mild epidemic form in Vienna at the end of 1916. Apparently, the same disease or variants of it appeared in Bucharest in 1915, in France in 1915-17, in Australia in 1917-18 ("Australian X-Disease"), in Japan in 1917 (the Japanese "B" type, apparently first identified in 1924), in London in the spring of 1918, in New York City in September, 1918, and in St. Louis and California and perhaps other parts of the United States in 1919. Probably unrecognized forms or variants of the same disease made their appearance much earlier, for example, in Copenhagen in 1615, in England in 1673, in Tübingen, Germany in 1712, in Italy in 1846 ("electric chorea") and at the time of the influenza epidemic of 1889-90 ("nona"), and in Japan in 1871.

² From a personal report from the Children's Hospital of Akron.

Near the beginning of the third decade of the present century, the disease had attained pandemic proportions, appearing in both sporadic (random, isolated) form and in large or small epidemics in many parts of the world. In England 5,039 persons were stricken in 1924; over 6,000 in Japan in the same year, with a mortality rate of 62 per cent, and over 5,000 in 1935, with a fatality of 42 per cent; 1,097 in St. Louis city and county in 1933, with a fatality rate of 20.1 per cent (an etiological agent was first definitely identified during this outbreak), and 338 in St. Louis city in 1937, with a fatality of 24.6 per cent (both St. Louis encephalitis). A number of minor epidemics have occurred in the United States, such as those in California in 1937 (the St. Louis type and the Western type of equine encephalomyelitis whose reservoir is infected horses), in Minnesota in 1937 (Western equine type), and in Massachusetts and Rhode Island in 1938 (38 cases with the Eastern equine type). In the 1948 epidemic in Japan the "Far East encephalitis" affected 4,707 "confirmed cases," with a mortality of 2,608, according to a personal communication from Crawford F. Sams, Brigadier General, Medical Corps Chief, stationed in the Japanese area. Although the epidemic forms have been of rare occurrence during the last decade, there will be no guarantee against recurrent outbreaks until effective preventives have been discovered.

Types of encephalitis³

A multiform neurotropic disorder. The investigations of the last two decades seem to show that there may be eight or

³On types of encephalitis consult Webster, Leslie T., "Classification of Primary Encephalitis of Man According to Virus Etiology," *Journal of the American Medical Association*, June 28, 1941, 2840-2841; and Neal, Josephine B., et al., *Encephalitis, A Clinical Study*. New York: Grune & Stratton, 1942. The interested reader is referred to this comprehensive volume for the references and the supporting data for many of the statements made in this chapter and for a detailed exposition of the neuropathology of this disorder, which, because of its technical nature and lack of space, is here omitted. See also Hassin, George B., *Histopathology of the Peripheral and Central Nervous Systems*. Baltimore: The Williams & Wilkins Company, 1938, pp. 197-202.

ten different kinds of this multiphase disorder, differing more or less in mode of origin, type of onset, severity, duration, detailed symptomatology, seasonal incidence, maximum age of susceptibility, and permanent postencephalitic residuals. The majority of identified strains are caused by some sort of ultramicroscopic organism, apparently of low degree of infectiousness (the disease rarely affects two siblings), classified as filtrable viruses because they pass through filters that will not transmit ordinary bacteria. Viruses are parasitic in nature, feeding exclusively on the living cells of their host. They are very resistant to temperature changes and germicides. Laboratory experiments have shown that only a limited number of animals are susceptible to a given virus. Viruses that have a special affinity for the central nervous system are called neurotropic. Among such viruses are those that cause rabies (isolated in 1881), infantile paralysis or poliomyelitis (isolated in 1909), and encephalitis.⁴

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Virus diseases—

Delbruck, Max and Mary, "Bacterial Viruses and Sex," *Scientific American*, November, 1948, 47-51 (a brief summary, stressing particularly sexual productivity among viruses).

Seiffert, Gustaf, *Virus Diseases in Man, Animals, and Plants*. New York: Philosophical Library, 1947.

The virus origin of encephalitis can be established by the isolation of the virus from the brain, blood, or spinal fluid of the patient, by intracerebral inoculation with infected brain tissue, and by the discovery of neutralizing antibodies (agents produced in the body that counteract injurious substances) in the serum during recovery. Of the encephalitis strains thus far proved to be of virus origin, only the St. Louis

⁴ Influenza, mumps, and measles are virus diseases, but the infections are not neurotropic.

and equine forms (the latter affecting both horses⁵ and human beings) are found in America.

The virus in the St. Louis type, which occurs in the late summer and early fall and affects adults particularly, has been found to be fatal to mice and pathogenic but not fatal to the *Macacus rhesus* monkey. Studies are filled with discussions regarding the mode of transmission, but recent investigations point to the common American mosquito (*Culex*), the brown mosquito (*Culex pipiens*), and chicken mites as the carriers (vectors).⁶ These insects feed predominantly on chickens, which are assumed to be the reservoir. The fatality for this type has varied from 20 per cent to 30 per cent.

The virus of the equine types, which affect young people particularly and which also appear in the summertime, is virulent to rabbits, guinea pigs, mice, rats, monkeys, and sheep. A variety of birds apparently supply the reservoir, such as the ring-necked pheasant, blackbird, cowbird, dove, hawk, and sparrow, and also the chicken, goose, and guinea fowl. Laboratory experiments have shown that several varieties of mosquitoes can serve as vectors for both the Eastern and Western strains. The wood tick serves both as reservoir and transmitting agent of the Western type, the milder form. The case fatality for the Eastern equine type in the Massachusetts outbreak was 60 per cent and for the Western equine type in the Saskatchewan epidemic in 1938, 12 per cent.⁷

The Japanese B type, occurring in the late summer and early fall, the counterpart of the St. Louis variety, from which it is indistinguishable epidemiologically although it is more

⁵ Over 184,000 horses were affected with equine encephalomyelitis in 39 states in 1938, about 20 per cent with fatal results. In 1939 after the introduction of the chick-embryo tissue vaccine the number was only 8,008.

⁶ Hammon, W. McD., and Reeves, W. C., "Recent Advances in the Epidemiology of the Arthropod-Borne Virus Encephalitides," *American Journal of Public Health and the Nation's Health*, October, 1945, 994-1004.

Smith, Margaret G., Blattner, Russel J., and Heys, Florence M., "The Isolation of the St. Louis Encephalitis Virus from Chicken Mites (*Dermanyssus Gallinae*) in Nature," *Science*, October 20, 1944, 362-363.

⁷ Neal, *Encephalitis, A Clinical Study*, p. 61.

fatal, is highly virulent for monkeys, mice, and sheep. It is probably transmitted by insects (especially mosquitoes, which may also act as reservoirs) from animals. The fatality and incidence of Japanese B are greater for adults than for children. This is also true of the St. Louis type.

The Russian type, which breaks out in the spring and summer (hence called vernoestival) and is endemic in forest areas, is pathogenic for monkeys and white mice and slightly so for guinea pigs. This disease is transmitted by ticks probably from rodents.⁸

A recent classification, based on a prolonged study of 50 cases, provides a tripartite grouping: (1) the virus groups, produced by six different virus diseases—such as poliomyelitis, lethargic encephalitis, influenza encephalitis, and measles encephalitis; (2) the toxic groups—such as diphtheria, pneumonia, scarlet fever, and meningo-encephalitis; and (3) traumatic types.⁹

Nervous involvements of common infections. Various neurological complications accompany various infectious diseases, such as measles, German measles (rubella), whooping cough, chicken pox (varicella), mumps, scarlet fever, pneumonia, smallpox, and vaccination against smallpox and rabies. Some of these neural complications resemble the acute or chronic symptoms of encephalitis, most frequently in connection with measles and vaccination against smallpox. These complications perhaps result from the activation by the infective agent of a latent encephalitic virus, or from a virus connected with the acute infection which becomes active because of lowered body resistance, or from the symbiotic action of two or more infections, or from some other factor.

⁸For a summary of the various characteristics of these and other special types of encephalitis see *ibid.*, pp. 1-133.

⁹Lurie, Louis A., Greenbaum, J. Victor, Leichtentritt, Bruno, and Rosenthal, Florence M., "Late Results Noted in Children Presenting Post-Encephalitic Behavior, A Follow-Up Study of Fifty Cases," *American Journal of Psychiatry*, September, 1937, 171-179.

Incidence

The disease attacks people of both sexes of all ages from infancy to old age (eighty-seven is the oldest reported age) in all walks of life in both hemispheres in the sporadic and epidemic forms. For a group of 8,000 cases culled from studies, the highest incidence occurred in the age range twenty to thirty, followed by ten to twenty (Neal). The onset for 80 per cent of 66 behavior encephalitics in the Child Guidance Home in Cincinnati, admitted between 1940 and 1946, occurred from five months to seven years, and before the age of seven years for 80 per cent of 113 acute cases examined in Cincinnati Children's Hospital and the General Hospital between 1919 and 1947. Sixty-one of 78 of the Guidance Home behavior cases were boys and only 17 were girls.¹⁰ The exact number of cases in the general population is unknown. Estimates are little better than conjectures because many of the milder, and even the more severe cases, are not recognized or reported. Neurologist Frederick Tilney estimated that only about 20 per cent of encephalitic cases are correctly diagnosed.

Symptomatology

It is difficult to outline in brief compass the chief mental and physical symptoms of the acute and chronic stages of the type that occurs on a global scale, the epidemic¹¹ form or the encephalitis lethargica of von Economo, of undetermined origin, because of the great variation in the range and severity of the symptoms in both the acute and the chronic stages and the vagaries of the course of the malady. Few patients fit into a fixed pattern. The clinical picture varies

¹⁰ Greenbaum, J. V., and Lurie, L. A., "Encephalitis as a Causative Factor in Behavior Disorders of Children: An Analysis of Seventy-Eight Cases," *Journal of the American Medical Association*, April 3, 1948, 923-930.

¹¹ This term, unfortunately, is a misnomer, as many of the cases occur sporadically rather than epidemically.

not only in different epidemics from time to time but also in different regional outbreaks. The variation in the clinical picture is doubtless due, at least in part, to the brain areas invaded by the pathogenic organisms. Any part of the encephalon seems to be vulnerable to this virus. Unpredictable remissions, exacerbations (flare-ups), and progressions may occur at any time during a period of years. Though many recover completely and permanently, others make only temporary recoveries.

The residuals in the chronic stage, whether mild or severe, may follow immediately after the acute phase or they may appear many years after apparent recovery; they may remain stationary or become progressively worse. It should, therefore, be clearly understood that not all the symptoms listed in the sections that follow are found in all patients.

Acute stage. The acute stage, sometimes ushered in very suddenly and sometimes very gradually, may begin with generalized complaints, such as headache, irritability, restlessness, malaise, dizziness, delirium, or with symptoms that are pathognomonic (specifically distinctive) of encephalitis, such as severe headaches, more commonly in the posterior region, somnolence lasting for days or weeks, fever of varying degrees, oculomotor disturbances (diplopia or double vision, frequently; dilated, contracted, or unequal pupils; strabismus; and palsy of the accommodation muscles), vertigo, sleeplessness, inversion of the sleep rhythm (somnolence in the daytime and insomnia at night, with excitability, noisiness, or maniacal behavior), myoclonic or choreic twitchings, motor restlessness, or paralysis of many muscles controlling speech, swallowing, the use of the hands or feet, and other activities, and Parkinsonism (see p. 404). Other possibilities include epileptic seizures (infrequently), loss of sensitivity (anesthesia), numbness, paresthesia (perverted sensations, such as tingling, prickling, or crawling sensations), or hyperesthesia. Over 90 per cent of Charles E. Gibbs's 144 enceph-

alitic children (behavior cases) under the age of fifteen in the special colony for such children in the King's Park State Hospital in New York between 1920 and 1929 had acute delirious or lethargic onsets followed immediately by impulsiveness, hyperrestlessness, and emotional instability.

The acute stage may vary in duration from about two to twelve weeks. The percentage of mortality and chronic sequelae are very high. The death rate reported in 1928 for 3,558 cases in England and Wales was 33.5 per cent. The percentage of recoveries among chronic epidemic encephalitics is given as low as 30 per cent by von Economo (Austria); and as high as 60 per cent by Felix Stern (Germany), even when the abortive cases and the intensively treated cases are eliminated.¹² Sixty-six per cent of complete recoveries were reported for the St. Louis 1933 cases after a period of three years, 11 per cent had muscular tremors suggestive of Parkinsonism, and 6.3 per cent were totally disabled.¹³ Of August Wimmer's 25 cases, 11 had died, 2 could not be located, and 12 developed serious psychiatric difficulties. Of Gibbs's 144 child encephalitics, 10 had succumbed, 8 had been discharged as cured (none having had any marked neurological signs), and 84 remained under treatment for the residuals. Of the others, 7 per cent had completely recovered, 50 per cent had developed Parkinsonism, the most dreaded complication, and 43 per cent were subject in some degree to emotional instability, behavior disorder, or delinquency. In the space of one year Morris Grossman's percentage of "improved" cases declined from 70 to 33 per cent.¹⁴ In 1924 R. J. L. Kennedy reported improvement in 42.5 per cent of 51 Mayo Clinic cases. Of the 40 cases for whom information could be obtained in 1940, 17.5 per cent were recorded as improved. This apparently includes estimates for those who

¹² Neal, *Encephalitis, A Clinical Study*, p. 202.

¹³ *Ibid.*, p. 13.

¹⁴ The foregoing data are culled from *ibid.*, pp. 342, 364 f.

had died. Of the 21 still alive 24 per cent were listed as improved.¹⁵ Only 4.9 per cent of Holt's 162 encephalitics "with sequelae" examined at the Boston Psychopathic Hospital were reported as "complete recoveries" after from 10 to 17 years. Only one complete recovery occurred among 110 cases of Parkinsonism. Of 129 with sequelae still alive, 22 were reported to be self-supporting. Encephalitis was the direct cause of 49 deaths. The fewest deaths occurred among those with behavior disorders, but they were the most incapacitated socially. Of 43 "previously showing behavior disorders," only 25.6 per cent were completely recovered or were employed and self-supporting after a period of ten years.¹⁶

In the first study of 50 encephalitics, 39 boys and 11 girls, admitted to the Child Guidance Home of the Jewish Hospital in Cincinnati because of behavior and personality disturbances, most of them years after onset of the conduct difficulties, only 9 had become adjusted during a follow-up period of from 4 to 26 years (most of them for 15 years), although the majority had received the "best available medical, psychiatric, and social therapeutic treatment." Twenty-seven were classified as virus cases, 14 as toxic, and 9 as traumatic types. The age of onset for 50 per cent of the group varied from five months to five years, although no history could be found for "an attack of encephalitis" in the "great majority of cases." The behavior and personality disorders followed the acute stage immediately in 36 cases. The abnormal behavior was grouped under three categories: (1) simple behavior disturbances; (2) psychopathic behavior; and (3) psychotic behavior. All except nine also manifested neurological disturbances, such as ocular disorders, seizures, or Parkinsonism. It should be borne in mind that these results

¹⁵ Heersema, Philip, "Prognosis in Postencephalitic Disorders," *American Journal of Diseases of Children*, October, 1940, 783-798.

¹⁶ Holt, William L., "Epidemic Encephalitis, A Follow-Up Study of Two Hundred and Sixty-Six Cases," *Archives of Neurology and Psychiatry*, December, 1937, 1137-1144.

are based upon the study of a selected group, referred because of conduct disorders.¹⁷ In the second study, which includes 28 additional cases, only 12 had "made a fair social adjustment" (8 virus cases, 3 toxic, and 1 traumatic). The unadjusted cases showed a larger ratio of toxic and traumatic forms, psychopathic and psychotic cases, and cases of intellectual deterioration.¹⁸

Chronic encephalitis (postencephalitis). The main interest of the psychologist, educator, and social worker is in the chronic stage if the patient becomes a candidate for special educational rehabilitation and social care, as is often the case. The severity of the chronic defects, especially the motor ones, does not always bear a close relation to the severity of the acute attack. In fact, the symptoms of chronic encephalitis often develop in the absence of any acute stage. Thus, 149 of Neal's 507 cases had no history of an acute attack.¹⁹ The chronic stage may follow the initial onset at once or it may be delayed a decade or more, possibly indicating that the virus may have remained latent in the organism all the while. The development of the chronic stage is essentially insidious and progressive, but sudden outcroppings of exaggerated characteristics are liable to occur without warning. The rate of progression differs greatly with individuals and is marked by periods of remission, intermission, or improvement. Improvement rarely occurs spontaneously. It occasionally results from appropriate medical treatment. Many aberrant symptoms are transient and recurrent. The chronic sequelae include a multiplicity of motor abnormalities, some due to the infection of the basal organs of the brain, which develop most frequently when the initial attack occurs during adulthood or late adolescence, and a multitude of behavior and personality disturbances with or without intel-

¹⁷ Lurie, Greenbaum, Leichentritt, and Rosenthal. "Late Results Noted in Children Presenting Post-Encephalitic Behavior."

¹⁸ Greenbaum and Lurie. "Encephalitis as a Causative Factor," p. 927.

¹⁹ Neal, *Encephalitis, A Clinical study*, p. 217.

lectual deterioration, particularly prominent when the onset occurs in childhood.

Motor abnormalities. These include a host of inconstant motor disorders and the Parkinsonian syndrome,²⁰ the most prominent among the motor disorders. The latter, which



FIG. 20. Propulsive gait in postencephalitic Parkinson disease. The patient may walk with head and body sharply inclined forward as if about to fall.*

rarely develops before puberty, includes, chiefly, muscular rigidity, coarse tremors (less common than those in paralysis agitans, or shaking palsy of older people, which are accompanied by pill-rolling), and slowness of movements (bradykinesis). The growing rigidity, as well as the concomitant but perhaps independent tremors which are accentuated when attention is directed toward them, manifests itself in the increasing inability to execute fine coordinated finger movements (shown, for example, in writing disturbances, including the writing of small, irregular characters); in speech disorders (slow, weak, high-pitched, monotonous, indistinct speech, sometimes abbreviated speech, mutism, or stuttering); in the expressionless, immobile, mask-like facies often accompanied by wide palpebral fissures and

²⁰ For a recent summary see Heath, James W., "Clinopathologic Aspects of Parkinsonian States; Review of the Literature," *Archives of Neurology and Psychiatry*, October, 1947, 481-497.

* From Major, Ralph H., *Physical Diagnosis* (3d edition). Philadelphia: W. B. Saunders Company, 1945, p. 32.

a reptilian stare; and in a stiff and eventually stooped posture and disturbances of walking. The gait may become unsteady, and the steps slowed down and shortened. At times the patient may rush forward with head and body inclined as if he were falling (propulsive or festinant gait), as shown in Fig. 20. He may also walk backward (retropulsion). Eventually he may become bedfast because of the paralysis or stiffness of the leg muscles. A host of hyperkinetic phenomena may be noticed, such as tics or spasms of many muscle groups (the eyelids, facial muscles, the abdominal or chest muscles), spasmodic coughing, snorting, choreic or athetoid movements, movements of the tongue, eye movements and fixations ("oculogyric crises"), and muscle contractions (for instance, wry-neck). Other disorders include sluggish pupil responses, disturbances on the vegetative level (temperature changes, excessive perspiration and salivation, irregular breathing spells—slow, quick, or inhibited breathing), and Parkinsonian disturbances of the sleep rhythm, excessive accumulation of fat (sometimes of the Fröhlich type), sex disturbances (precocious or delayed puberty, excessive or deficient libido), and headaches and other pains.

ILLUSTRATIVE CASE: postencephalitic Parkinsonism. J. A. H., the son of a paper hanger and painter, suffered an attack of encephalitis at the age of 12-11 which, according to his own handwritten "life story," was preceded by an eight-week attack of "influenza" many months before, not recorded in the anamnesis (past history of a disease). According to his story the encephalitic attack began when "everything went black" as he was on his way to get some cotton from a cotton mill near-by. As he regained his sight everything "looked like two" (diplopia). He spent six weeks in bed, asleep for three weeks. His school work and conduct prior to the attack were reported to be "very good." But he was "never bright" after the attack and was unable to "study much." He "sat around" instead of playing with children, as he had done before. He was the seventh child of 13 siblings; all of the other seven alive at the time were "normal and well adjusted." At the age of seventeen he was sent to the Caswell Training School in North Carolina. On entry the signifi-

cant physical findings were: some muscular rigidity, poor motor coordination, a "dragging" gait, and a speech impediment.

The summary sheet at the age of twenty-two (apparently when the psychological service was inaugurated in the institution) indicated that his physical condition had grown steadily worse for two or three years. He walked very slowly and stiffly, dragging his left foot and extending his arms stiffly forward. His mouth was open, his tongue protruded, he drooled occasionally, his eyes had a glassy stare (reptilian stare), his stiff fingers were subject to a slight tremor, his movements were very slow, and his speech indistinct and delayed. Nevertheless, he was very cooperative, responsive, and attentive, and "twisted his tongue" in the determined effort to enunciate more clearly. He was subject to spells of depression because of his desire to go home and the apparent hopelessness of his condition, and was sensitive about his physical abnormalities because the boys made fun of the way he walked. He would not "mix with them."

The report for the next two or three years indicated improvement from the use of stramonium. He reacted more promptly, walked better, replied more clearly, the muscles became less stiff, the face more expressive, the jaws closed better, and he became happier. Concurrently he received training from the psychologist designed to improve his speech, to hold the jaws closed, and to overcome lack of confidence. At his request he was again enrolled in the school for work in arithmetic, geography, and history. He was dependable, worked well without supervision in the cottages, and got along well with the other boys. Withdrawal of the drug, however, caused reversion to his former state.

The results of the psychological tests at various ages are as follows:

<i>Life Age</i>	<i>Stanford-Binet</i>		<i>Porteus Maze</i>		<i>Form I. Binet</i>	
	<i>Binet Age</i>	<i>I. Q.</i>	<i>Porteus Age</i>	<i>I. Q.</i>	<i>Binet Age</i>	<i>I. Q.</i>
22*	12-3	77	12-6	89		
24-3*	11-8	73	13-6			
28-11						
					12-0	80

* Goodenough drawing test age 13-0. The Healy Pictorial Completion Test II at 24-3 yielded an age of 20 plus. A copy of the institution's case record was made available by Miss Elizabeth Brown, psychologist.

The subject's progressive physical deterioration was not accompanied by a concomitant mental deterioration, as evidenced by test findings. His intelligence level remained about the same for over six years. According to the clinical notes he always showed good judgment and comprehension and was self-critical and anx-

ious to do his best, but he was always slow in his responses. He would rate not lower than backward or borderline in general intelligence on the basis of clinical findings.

When first seen by my group of Duke University students at the age of 25-6 after about two years on the stramonium treatment, the most conspicuous symptoms were the reptilian stare and the fixed forward gaze as he walked slowly and rather stiffly across the floor. He responded willingly and talked intelligently and fairly distinctly about his affliction. When last seen (in the cottage, not at the clinic) at the age of 29-6 he had undergone marked physical and mental deterioration. His hands were paretic, his legs stiff, and he walked very slowly and unsteadily across the floor. His eyes were turned fixedly upward toward the ceiling, the mouth was open, the tongue protruded and moved in and out, and it was impossible to elicit any response to questions or any recognition. He died the following year (1942) at the age of 30-8, apparently a potentially normal person, a victim of a brain virus.

Behavior and personality disorders. These disorders, as well as intellectual deficiencies, are more likely to develop when the onset occurs in infancy or early childhood in severe forms. Although these early-life cases afflicted with psychic sequelae are not entirely free from neurological complications, the motor disorders ordinarily are less prominent. The somatic disturbances with which they may be troubled include nightmares, sleep reversals, disturbances of respiration, tics, seizures, athetoid or choreiform movements, great restlessness, and even Parkinsonism in varying degrees of severity.

The range and severity of the personality anomalies differ from person to person, but the major disorders include emotional instability, suspiciousness, unreliability, excessive restlessness, aggressiveness, quarrelsomeness, abusiveness, assaultiveness, disobedience, meddlesomeness, impudence, vaingloriousness, egocentrism, lack of inhibition, and hyperimpulsiveness. Although the malbehavior of encephalitics, as with normal children, may represent efforts to get into the limelight or to evade disagreeable responsibilities (an escape

or defense mechanism), many conduct disorders are largely of an uncontrollable, impulsive character. The all-over picture is one of hyperkinesis. Children who have previously been amiable and well mannered may suffer sudden and profound character alterations. Sometimes the picture is one of abrupt and apparently purposeless outbursts of violence. Temperamental outbreaks may occur with dramatic suddenness without provocation. Ungovernable impulses of the moments account for a great variety of behavior disorders: the use of obscene language; streaks of assaultiveness, such as hitting, kicking, biting, throwing missiles, spitting at persons; the destruction of property; setting fires; stealing; lying; hypersexuality and committing sex offenses; running away, and the like. These children often realize what they do, but cannot explain why they misbehave, and often seem powerless to control their action. Although often expressing deep remorse when reprimanded, they may immediately repeat the offense. In some children the disease merely aggravates pre-existing delinquent trends or perverse conduct. Organic lesions probably explain why the infliction of punishment or the attempt to reason with impulsive encephalitic miscreants is usually ineffective. In fact, repressive measures often aggravate the condition. The excessive impulsiveness or lack of control of the primitive urges may be related to structural degenerative changes in the thalamic or hypothalamic area at the base of the brain, functionally related, perhaps, to the individual's primitive impulses and emotions. Some conduct disturbances that have followed in the wake of measles, scarlet fever, diphtheria, rheumatism, or influenza, sometimes with choreiform movements, may well represent the residuals of an undetected virus infection of encephalitic character. A knowledge of the etiological background is essential as a basis for skilled medical, psychological, psychiatric, and educational treatment of all such cases. Intelligent control must replace forcible restraint.

Many postencephalitics are hampered by major and minor psychopathological disturbances of the organic reaction type.

Such aberrant functioning includes states of inattentiveness, apathy, catatonic stupor, withdrawal, depression, anxiety, hypomania, delirious and confusional episodes, compulsions, hallucinations, dissociations, delusions, and the like. Many encephalitics with psychiatric disorders possess considerable insight regarding their abnormal behavior. A. C. Parsons reports that 27 per cent of 1,776 chronic encephalitics evidenced "symptoms of outstanding mental disorder." Of 174 patients reported by Cooper, 98 manifested depression, 5 confusional behavior with hallucinations, 10 convulsive attacks, and many showed various behavior disorders.²¹ Of the 78 behavior encephalitics in the Cincinnati Guidance Home, 46.1 per cent were diagnosed as psychopathic and 20.5 per cent as psychotic (the latter including children who were slovenly, uncontrollable, moody, hallucinatory, negativistic, refusing to wash or bathe, indulging in irrelevant talk, and the like).²² A continuance in the chronic stage of the mental symptoms, presumably both major and minor ones, was found in 53.6 per cent of Wilson's cases that showed the mental abnormalities in the acute stage, and 14.9 per cent of other patients developed mental symptoms later. Although only 4.4 per cent of Albert A. Rosner's 201 cases were "permanently psychotic," the percentage is raised to 12.8 if the behavior cases are included and 27.3 per cent if those subject to "affect disorders" and severely retarded non-psychotic adults are also added. Of 3,350 notified cases of encephalitis in England between January, 1919 and December, 1922, only 0.8 per cent could be certified as insane. Of over 4,000 behavior children under fifteen at the time of the first observation in the Psychiatric Division of Bellevue Hospital from 1934 to 1940, only 55 were encephalitics, or 0.14 per cent.²³ Of 2,700 of Greenbaum and Lurie's unselected children "who presented all types of behavior disorders," 2.9

²¹ Cooper, H. A., "The Mental Sequelae of Chronic Epidemic Encephalitis and Their Prognosis," *Lancet*, 1936, 2:677-679.

²² Greenbaum and Lurie, "Encephalitis as a Causative Factor," p. 925.

²³ The above figures are quoted from Rosner's chapter in Neal, *Encephalitis, A Clinical Study*, pp. 326 f.

per cent were encephalitics. This, of course, does not furnish any clue as to the proportion of encephalitics subject to conduct disorders. Although wide differences exist in the available reports, only a small ratio of the total number of encephalitics can be classified as actually psychotic or can be certified as insane.

ILLUSTRATIVE CASE: an encephalitically produced behavior disorder. Jo was referred in 1929 to the writer's psychoeducational clinic in Baltimore by an officer of the Juvenile Court because of the theft of a bicycle. He had been haled into court once before because of some minor infraction. His Stanford-Binet I. Q. at the time was about 85. Investigation revealed that he had been a model pupil in one of the city elementary schools and had been regularly promoted until he suffered some infectious attack (about two years earlier), later diagnosed as encephalitis. Although this attack left him without any patent neurological complications, the quality of his school work deteriorated and he became restless and subject to minor conduct disorders. The recommendation to transfer him from the irritating, noisy, bustling city environment in which he lived to the home of a relative and to a small school taught by a stable, understanding teacher in a quiet rural area enabled him to make a satisfactory social adjustment, according to a follow-up report received several months later.

Intelligence arrest or deterioration. The data from the numerous investigations of encephalitics by means of clinico-objective tests, usually the Binet, are somewhat conflicting. In the ten studies tabulated by Pinter, Eisenson, and Stanton,²⁴ the mean or median I. Q.'s based on different versions of the Binet ranged from 73 to 91 and the individual diagnoses from idiocy to mental superiority. The rating on a second test at various later intervals was lower in most instances. D. Paterson and J. C. Spence reported a state of "permanent and hopeless idiocy" for 7 of 25 children (28 per cent) examined in England who were intellectually normal before the onset of the attack. Twenty-five of 36 children (69.4

²⁴ Pintner, et al., *The Psychology of the Physically Handicapped*, pp. 294 f.

per cent) examined in the Mental Hygiene Clinic of Bellevue Hospital in New York were classified as "mentally deficient"; 11 were listed as morons, 4 as imbeciles, 2 as idiots, 4 as retarded, and 4 were unclassified. The I. Q. ranges (test used not disclosed) in the first Cincinnati group of behavior encephalitics was from 51 to 118. Two were classified as intellectually superior, 16 as average, 11 as subaverage, 7 as borderline, and 14 as feeble-minded. These classifications were probably based on the I. Q. distribution. Several years later on retests, 34 of 44 cases had deteriorated, one had improved, and 9 had remained stationary.²⁵ In the later summary of 78 cases, 3 were classified on admission on the basis of psychometric tests as superior, 26 as average, 17 as subaverage, 14 as borderline, and 18 as feeble-minded. Of 47 retested "several years after the child's dismissal" from the Home, 9 had the same I. Q., 1 had improved, and 37 had deteriorated. The prognosis of these cases—it must be recalled that they were behavior cases, with many psychopaths and psychotics—was declared to be "highly disappointing" in spite of the best available treatment.²⁶

The Stanford-Binet I. Q. range for 108 children, ages two to ten on the initial test, tabulated by Brown, Jenkins, and Cisler, was from 40 to 117,²⁷ with an average of 77. A decrease in I. Q. of over four points between the first and last test (at varying intervals up to many years) was shown by 44.4 per cent; 39.8 per cent remained approximately stationary; 15.7 per cent increased over four points. In this group no significant differences were found for those with behavior disorders or Parkinsonism. Possibly the age difference between this group and the Cincinnati cases may explain the differences in the findings. In the Brown-Jenkins-Cisler

²⁵ Lurie, Greenbaum, Leichtenstritt, and Rosenthal, "Late Results Noted in Children Presenting Post-Encephalitic Behavior," pp. 1018 f.

²⁶ Greenbaum and Lurie, "Encephalitis as a Causative Factor," p. 927.

²⁷ Brown, Andrew W., Jenkins, Richard L., and Cisler, Lillian E., "Influence of Lethargic Encephalitis on Intelligence of Children," *American Journal of Diseases of Children*, February, 1938, 303-321.

study the reduction in intelligence was found slightly greater for the children in whom the onset occurred before the age of ten. Some years earlier Mandel Sherman reported that the intelligence level of many children was not impaired when the attacks occurred before the age of six or seven, in spite of emotional and personality disorders.²⁸ Heershema found a decided likelihood of mental deficiency when the attacks occur before five.²⁹ Dealing with children under six years of age, Laurretta Bender and Helen Yarnell reported immediate cessation of mental growth in many cases at the stage when the attack occurred.³⁰ Frank R. Ford's conclusion is that the intelligence may be arrested in infancy but no marked reduction occurs between three and ten. On the other hand, Duncan's conclusion is that "nearly one-half of all the children up to the age of fourteen are impaired mentally or physically to such a degree that they cannot receive instruction as normal children."³¹ The intelligence level is doubtless underrated in children in whom muscular rigidity inhibits or retards their responses.

Obviously the deteriorating effect on intelligence differs greatly with individuals, dependent, perhaps, on the type of malady and the native resistance of the nerve cells. Some of the mentally deficient encephalitics are, doubtlessly, primary aments. Most of the subnormals are on the high grade defective, borderline, or backward levels. Some of the mentally deficient children tend to be placid, inert, indolent, and indifferent. They may be well-behaved except for occasional instabilities or infractions, and may exhibit few of the classical neurological signs of the disease. On the other hand, other mental retardates are restless, difficult to control, or delinquently inclined.

²⁸ Sherman, Mandel, and Beverly, B. I., "The Factor of Deterioration in Children Showing Behavior Difficulties after Epidemic Encephalitis," *Archives of Neurology and Psychiatry*, 1923, 10:329-343.

²⁹ Heershema, "Prognosis in Postencephalitic Disorders," p. 787.

³⁰ Neal, *Encephalitis, A Clinical Study*, p. 367.

³¹ Duncan, A. G., "The Sequelae of Encephalitis Lethargica," *Brain*, 1924, 47:91.

ILLUSTRATIVE CASE: a placid mentally defective encephalitic without conduct disorder. Ethel, a twin apparently of normal development, suffered an infectious attack at the age of two and a half, which produced temporary paralysis. The attack was later diagnosed as encephalitis by a Philadelphia specialist. She learned to walk, however, but continued to stumble and fall for years. No evidence of Parkinsonism ever appeared, but motor coordination remained poor. When referred to the writer's psychoeducational clinic at the age of 12-11, her Stanford-Binet age was 6-0, I. Q. 46. At 14-10 her Binet age had dropped to 5-2, and the I. Q. to 36 (however, on a different Binet version, Form L). On the Wallin-Gilbert (Cutsforth) Individual Attainment Scale her grade level was II— in reading and spelling, I— in arithmetic, and I+ in language. On the Arthur Performance Scale, Form I, she scored below intelligence age 5-6. On the Goodenough Drawing Test she obtained an intelligence level of 4-3, I. Q. 33. Coordination was poor; memory span and vocabulary were superior to comprehension and reasoning ability. She was admitted to the special class with the emphasis on concrete and practical training and the development of confidence and good health habits. The progress report at the end of the year (1936) indicated that her greatest weakness was poor muscular coordination and lack of interest in activities. She was rated poor in industry, attention, effort, persistence, initiative, and dependability, but good in disposition and behavior. "Most of the day she sits idle and quiet." She was occasionally subject to sudden impulses, such as throwing the toilet paper about in the school toilet or throwing clothes from the bureau drawer around in the room at home. She entered a private school in January, 1938 and transferred to another private school after a year, where she was still enrolled (in 1946) at the age of twenty-four. At twenty-four she could add but not subtract, possessed some ability in reading, had a "good memory, especially for telephone numbers," and remained in good physical condition free from obvious neurological symptoms.

Treatment

Medical. A great variety of forms of medical treatment has been used empirically for the purpose of immunization against the infection, for amelioration or cure of both the acute and the chronic symptoms, and for the improvement of

the general health and morale of the patient, including drugs galore, vaccines, serums, surgery, physiotherapy, occupational therapy, hydrotherapy, and psychotherapy. The vast majority of drugs has proved of little value except as adjuvants in the control of certain annoying symptoms. The "F" vaccine (a herpes virus vaccine) and various alkaloids, especially "bellabulgara" (now available in tablets) made from the roots of the Bulgarian belladonna plant, have apparently proved of most value for the removal of the motor abnormalities and for the general improvement of the patient. For a group of 93 cases treated in the acute stage with vaccine "F" ("X" proved more effective with some), Neal reports a mortality of 11.8 per cent as compared with 24.6 per cent for 293 cases treated without the benefit of the vaccine at the Division of Acute Infections of the Central Nervous System of the Health Department of New York City.³² Of 81 chronic cases under treatment from two to five years with vaccine "F," 58.0 per cent showed improvement, whereas the corresponding figure was only 7.6 per cent for 250 cases receiving symptomatic treatment without vaccine. The number who grew worse in the first group was only 13.6 per cent as compared with 72 per cent in the non-vaccine-treated group.³³ Of 85 chronic adult cases treated with bellabulgara for over two years, 36.5 per cent improved greatly, 51.8 per cent improved moderately, and 11.7 per cent only slightly.³⁴

It is important in the treatment of encephalitis to institute the therapy before permanent damage is done to the nerve cells. Suspicious cases should be referred without delay for competent medical diagnosis.

The relaxant drug, curare, is of value in relieving Parkinsonian rigidity, contractures, and pain from muscle tension.³⁵

³² Neal, *Encephalitis, A Clinical Study*, p. 277.

³³ *Ibid.*, pp. 283, 285.

³⁴ *Ibid.*, p. 297. For other studies, see pp. 302 f.

³⁵ Schlesinger, Edward B., "Recent Advances in the Use of Curare in Clinical Practice," *Bulletin of the New York Academy of Medicine*, October, 1946, 520-529. For a recent comprehensive treatise see McIntyre, Archibald R., *Curare: Its History, Nature, and Clinical Use*. Chicago: University of Chicago Press, 1947.

Other relaxant drugs include prostigmine and myanesin.³⁶ According to Berger, myanesin is without side effects and is superior to curare and several other relaxants in reducing muscle spasm, spasticity, rigidity, and tremors in cerebral hemiplegia and in diplegia, spastic, athetoid, and choreiform types of disease, and Parkinsonism. Success in muscle training is often contingent upon the removal or lessening of muscle rigidity.

Psychological and educational treatment. Because of the hyperexcitability of the nervous system, diplomacy of a high order is needed in the psychological handling of many encephalitics. They should be provided with placid, well ordered surroundings—in the home, school, and hospital—free from friction, vexation, and excitement. The patient's morale must be constantly fortified against the disappointments and discouragements that go with the vicissitudes of the malady. The oversolicitous parents must be warned against the crippling effects of overprotection and coddling, which may hamper the child's attainment of emotional maturity or development of independence, self-reliance, initiative, and resourcefulness. Positive efforts must be put forth to develop initiative and resoluteness to fend for one's self. Unsympathetic and harsh parental martinets should be enlightened regarding the true nature of the child's impulsive outbreaks and should be cautioned against the use of disciplinary severities that will only accentuate the child's instability. Parents and teachers should understand that the encephalitic child's misbehavior in most cases is not wilful or intentional, but is largely conditioned by an organic brain disease. They must, at the same time, be cautioned against assuming an uncooperative, indifferent, fatalistic attitude toward the victims of this malady. Some can eventually be restored to acceptable social behavior by appropriate drug and vaccine therapy and environmental control. If the home is too disturbing, a

³⁶ Berger, F. M., and Schwartz, R. P., "Oral Myanesin in Treatment of Spastic and Hyperkinetic Disorders," *Journal of the American Medical Association*, June 26, 1948, 772-774.

quiet retreat should be found for the child during the acute and convalescent stages and sometimes during the chronic stage as well. Removal from the home may also be desirable when the child's impulsive, ungovernable, or violent conduct exerts an inimical influence on the other members of the family.

Evidence of the effect of a well regulated school and dormitory régime free from undue excitement and strain, with needed educational, psychological, and medical treatment adjusted to individual requirements, is supplied by the experiment conducted in the Franklin Special School of the University of Pennsylvania Hospital, established in 1925.³⁷

Other special facilities provided for encephalitics include the clinic established in Rome by Queen Elena in 1934 (with later branches elsewhere and with, it is alleged, 100 per cent of "virtual cures" of the mild cases with the use of bellabul-gara), the hospital opened in Germany in 1937 exclusively as a treatment center, and the divisions in the King's Park Hospital in New York and in the Allentown State Hospital in Pennsylvania.

The children committed to the Franklin School were all difficult behavior cases free from neurological complications and Parkinsonism and of fairly normal intelligence. Various régimes were tried experimentally until 1933 when the administration was unified and more extensive psychological and psychotherapeutic treatment was provided each child by psychiatrists—treatment interviews, play therapy, with or without interpretation, relationship therapy, re-education, and habit formation. Although the daily routine was restrictive, it was constructive, tolerant, and individualized. Of 85 children admitted during a ten-year period, 9 were still in the

³⁷ Bond, Earl D., and Appel, Kenneth E., *The Treatment of Behavior Disorders Following Encephalitis*. New York: The Commonwealth Fund, 1931.

For the successful handling of an encephalitic, who had previously been expelled from a regular grade, in a school for cripples see Cole, Blanche E., "The Problem of Social Adjustment Following Epidemic Encephalitis in Children," *Mental Hygiene*, 1924, 977-1023.

school. Of the 76 discharged, 20 or 26 per cent had been successfully restored to their pre-encephalitic condition. However, many of 56 who had become quite tractable in the school had a "poor" home record, because of adverse environments. Many children changed remarkably for the better in school and then "broke down under the stress of alcoholic fathers, infantile mothers, criminal neighborhoods." ³⁸

During the acute and convalescent stages, the primary, if not the exclusive, consideration is medical treatment and rest and relaxation. The program of motor re-education and academic training should be deferred until the nervous condition has been largely stabilized. Motor disabilities can be mitigated or overcome and motor coordination can be improved by a combination of medical treatment, formal physiotherapeutic treatment (massage, active and passive movements, corrective gymnastics, thermotherapy, hydrotherapy, radiation), occupational therapy, and craftwork. All such work must be adjusted to the stage of recovery and strength of the individual and must be interspersed with needed rest periods. The program of rehabilitation should include play and music therapy; socialization through cooperative recreational and work projects and through active participation by those who are ready for it in the curricular or extracurricular activities of the school (entertainments, general assemblies, club activities, and so on); individual psychotherapy, counseling, vocational guidance, and placement.

Properly treated, many encephalitics of appropriate age are admissible to the regular grades or special classes (for the mentally retarded) in the public schools. The exceptions are the children of idiot or lower imbecile levels and the unstable, disturbing cases for as long as they remain unmanageable. The lowest grade cases and those whose irrational, impulsive behavior aberrations create serious disturbances in

³⁸ Bond, Earl D., and Smith, Lauren H., "Post-Encephalitic Behavior Disorders, a Ten-Year Review of the Franklin School," *American Journal of Psychiatry*, July, 1935, 17-33.

the classroom require institutional or colony treatment and care. It must be borne in mind, however, that only a few residential institutions or annexes exist that are specifically designed for encephalitic children. Classrooms in which the atmosphere is too competitive, tense, and boisterous are not suitable for hair-trigger types of children. Some of the more hyperexcitable ones would respond properly in a small group in a special class, provided the class is taught by a stable, tolerant teacher who knows how to establish rapport and get willing compliance.

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For a biographical account of the disease over a period of 30 years see "Effects of Encephalitis Lethargica," *Lancet*, 1948, 904-905.

Chapter 17

THE ORTHOPEDICALLY DISABLED: CEREBRAL PALSY¹

No other kinds of handicapped children have aroused a greater degree of popular solicitude than those subject to muscular paralysis and bone disorder. This widespread interest is due to a number of factors, the most important of which are listed below.

First, the obtrusive nature of most severe degrees of muscular palsy and the grave impediment they create for the child's successful educational and socio-economic adjustment.

Second, the widespread prevalence of muscular, bone, and joint disabilities. In the table supplied by the subcommittee on the crippled child of the committee on special education of the White House Conference on Child Health and Protection² the number of crippled children under eighteen reported from different places in the United States varies from 0.10 to 11.3 per 1,000 of the general population and from 2.96 to 16.67 per 1,000 of the school population. The wide variation is probably due chiefly to differences of definition depending upon the purpose of the surveys. The inclusion of cardiopathic children, in accordance with the present trend, would greatly increase the number. Thus the proportion reported for two counties in Michigan in 1930 inclusive

¹ This chapter has been read by George J. Boines, M.D., Director of the Wilmington, Delaware, Kenny Clinic, to whom gratitude is expressed for a number of revisions.

² *Special Education, the Handicapped and the Gifted*. New York: Century Company, 1931, pp. 24 f.

of cardiacs was 26 per 1,000 among 8,000 school children. An estimate of from 2 to 4 crippled children for each 1,000 of the general population would seem to be conservative. The conservative estimate of the White House Conference Committee, based on its inquiries, was that "there are more than 300,000 crippled children in the United States, and that at least one-third of these are in need of special educational opportunities."³ The number of crippled children of all kinds on the state registers, including Alaska, District of Columbia, Hawaii, and Porto Rico, was 404,602 in 1945, according to the tabulation issued by the Federal Children's Bureau on September 9, 1946.

Third, the effective promotional work by many influential organizations, such as the Rotarians, the Kiwanians, and Shriners, the National Society for Crippled Children and Adults (founded in 1921 by "Daddy" Allen as The International Society for Crippled Children), many State Societies for Crippled Children, and the National Foundation for Infantile Paralysis (founded in 1938 by Franklin Delano Roosevelt) with its numerous chapters in every state (2,728 in 1947) and its annual nation-wide March of Dimes drives.

Fourth, the liberal federal subvention for programs of rehabilitation of the crippled. This program began in 1918 in connection with the vocational rehabilitation of disabled soldiers, sailors, and marines. The scope of this program has been greatly enlarged since World War II. In 1920 the vocational rehabilitation act was passed for the vocational rehabilitation of disabled civilians of employable age. This program, administered by the states on a matching basis, under the general supervision of the Office of Vocational Rehabilitation of the Federal Security Agency (as a part of the Social Security Act since 1935), has been greatly extended from time to time, with generously increased grants, to include almost all kinds of handicapped persons of "employable age" (usually from sixteen to an indefinite upper limit) for

³ *Ibid.*, p. 32.

almost any kind of service or appliance needed for correcting or mitigating any "static disability" (that is, an arrested or relatively permanent and unprogressive disability) which "constitutes a substantial handicap to employment," provided "such correction or modification should eliminate or substantially reduce such handicap within a reasonable length of time" (Public Law 113, 1943). The 1943 revision of the law includes the mentally handicapped for the first time. The U. S. Children's Bureau, established in 1912 (and transferred in 1946 from the Department of Labor to the Federal Security Agency), established its crippled children's division in 1935, which was consolidated in 1941 with the maternal and child health division into the Division of Health Services. Grants-in-aid were first made available in 1936 to the states for services for crippled children.

Most prevalent types

Although only a minority of the large group of orthopedically disabled children constitute mental problems from the standpoint of intelligence deviation, any text on mentally deficient children and retarded children would be seriously deficient without a section on crippled children. Many of them, irrespective of intelligence level, create a variety of special education problems whether they are registered in the regular grades or in special classes or schools. Because of the current nation-wide interest in child victims of cerebral palsy and poliomyelitis (infantile paralysis) most of the discussion is confined to these two groups, which represent the most prevalent types of orthopedically handicapped children. This is shown by the classification as of December 31, 1944, by the Federal Children's Bureau of 341,022 crippled children on the state registers. The largest categories are those of poliomyelitis and cerebral palsy, constituting 18.3 per cent (62,373 cases) and 9.8 per cent (33,380 cases), respectively, of the entire group. Undoubtedly, the children reported do not include many of the subclinical types of polios and many

homebound cerebral palsy cases may remain unreported or concealed because of family pride or ignorance. Of all the orthopedically disabled, children with cerebral palsy have been the most neglected until recently, from both the medical and the educational point of view, especially, perhaps, those of normal mentality who, misunderstood and underrated, have been kept at home deprived of educational facilities and social contacts. Many of the mentally deficient cases (and some of the misdiagnosed cases) have found refuge in institutions for mental defectives.

Age and sex incidence of orthopedic handicaps

According to the report of the White House Conference Committee on the crippled child, 83 per cent of 1,531 children under twenty-one years of age were crippled before the age of six. Seventy-nine per cent of the Wisconsin orthopedically disabled children were crippled before the age of seven.⁴ There is a preponderance of boys, varying in different cities from 2.4 per cent to 9.6 per cent.⁵ Among 1,845 children under twenty-one with cerebral palsy known to the Crippled Children's Division of the Department of Health of New York City, the preponderance of males amounted to 14.2 per cent.⁶ Of the first 500 cerebrally palsied children studied by the New Jersey State Crippled Children's Commission, the excess of males amounted to 16 per cent.⁷

Classification of children with neuromuscular disabilities

All degrees of paralysis exist from a barely perceptible crippling or weakening of one muscle (the slighter degrees are

⁴ *Ibid.*, pp. 26 f.

⁵ Baker, Harry J., *Introduction to Exceptional Children*, p. 151.

⁶ *Orthopedically Handicapped Children*. The Committee for the Study of the Care and Education of Physically Handicapped Children in the Public Schools of the City of New York, The Board of Education, 1941, p. 79.

⁷ McIntire, J. Thomas, "The Incidence of Feeble-Mindedness in the Cerebral Palsied," *American Journal of Mental Deficiency*, April, 1946, 491-494.

referred to as paresis) to almost total paralysis, or the loss of muscle strength (myasthenia), or loss of the voluntary ability to move one or many muscle groups. The different varieties of dyskinesia (disordered motility) and hypokinesia (diminished motility) and of the often associated hypertonicity or hyperkinesia (excessive motility) that constitute constituent elements of the symptom complex of the paralyzes are as multiform as the varying degrees of severity of the different disorders.

Crippled children may be classified from the point of view of eligibility for state aid for special education services (usually assignment to special schools or classes) without reference to causation; or they may be grouped etiologically according to the pathogenesis (nature of the causative factor) or the nature and seat of the tissue destruction (referred to as pathology).

Educational definitions drawn in conformity with specific administrative regulations. The following definitions are based on practical educational considerations rather than on scientific groupings.

New York education law: a crippled child is one "under twenty-one years of age, who, by reason of physical defect or infirmity, whether congenital or acquired by accident, injury, or disease, is or may be expected to be totally or partially incapacitated for education or for remunerative occupation, but shall not include the deaf or the blind."⁸

The subcommittee on the crippled of the White House Conference on Child Health and Protection: "The crippled child, in the orthopedic sense, is a child that has a defect which causes a deformity or an interference with normal function of the bones, muscles, or joints. His condition may be congenital or may be due to disease or accident. It may be aggravated by disease, by neglect, or by ignorance."⁹ This definition is more specific. "Children are eligible for special

⁸ *Special Education*, White House Conference Report, p. 23.

⁹ *Ibid.*, pp. 23 f.

classes if they cannot attend regular classes with safety and profit to themselves and others.”¹⁰

Wisconsin Crippled Children's Division: “A crippled child is an individual under twenty-one years of age whose activity is so far restricted by loss, defect, or deformity of bones, or by affections of the neuromuscular system, as to reduce his normal capacity for education or self-support.”¹¹ Cardiacs recommended to orthopedic schools are also included.

Commission for the Study of Crippled Children of New York City: “A crippled child is an individual under twenty-one years of age who is so handicapped through congenital or acquired defect in the use of his limbs and body musculature, as to be unable to compete on terms of equality with a normal individual of the same age.”¹²

All the definitions above are broadly drawn with a view to the easy approval of a child for special class transfer. The following New York definition drawn for the purpose of limiting transfers to special orthopedic classes contrasts sharply with the earlier definition from the same area.

Subcommittee on Orthopedically Handicapped Children, Board of Education of the City of New York: “Principles of admission to special orthopedic classes should be rigid and only children in need of protective care or special educational methods which cannot be applied in the regular classes should be admitted to them.”¹³ “Only those unable to conduct their lives with safety to themselves or others should be admitted to special classes.”¹⁴ Under this concept from 3,900 to 5,500 of 19,400 were deemed proper special class candidates.¹⁵ It is obvious that definitions or classifications of this nature, although they may serve useful purposes, supply no

¹⁰ *Ibid.*, p. 41.

¹¹ *Care and Education of Crippled Children*. Madison, Wisc.: State Department of Public Instruction, Bulletin No. 4, 1937.

¹² *The Crippled Child in New York City*. New York: Commission for the Study of Crippled Children, 1940.

¹³ *Orthopedically Handicapped Children*, p. 11.

¹⁴ *Ibid.*, p. 19.

¹⁵ *Ibid.*, p. 20.

information regarding the type of case, the nature of the pathology, the symptomatology, or the pathogenesis of the disabilities. They possess no nosological value and do not convey any basic, scientific knowledge of the case.

Classification of paralysis according to the number of muscles involved. The paralysis may involve one limb or muscle group (monoplegia or local paralysis), one side of the body (hemiplegia), both sides (diplegia or quadriplegia), the lower extremities (paraplegia), or three extremities (triplegia, hemiplegia plus paralysis of one limb on the other side). (See Figs. 21 to 24.) The various types of specific paralysis known by different terms are too numerous for mention here.

Of Peterson's 451 cases, 332 were hemiplegics, 73 diplegics, and 46 paraplegics. Only one was a monoplegic.¹⁶ Among 146 noninstitutional "spastics" included in the New Jersey Birth Palsy Survey, the distribution was: hemiplegics, 16 per cent; paraplegics, 14 per cent; triplegics, 10 per cent; quadriplegics, 57 per cent; monoplegics, 1 per cent; unclassified, 2 per cent.¹⁷ In a later survey based on 500 cases the distribution was: quadriplegics, 56.6 per cent; hemiplegics, 25.6 per cent; diplegics, 9.0 per cent; triplegics, 6.0 per cent; monoplegics, 1.6 per cent; all others, 1.2 per cent. The age range of these cases was from one to twenty-four, median 9.8 years.¹⁸ The New Jersey distributions differ considerably from Peterson's figures.

CLASSIFICATION OF PARALYSES ACCORDING TO PATHOLOGY

Classification of paralyzes according to the location of the neuromuscular lesion. Organic paralyzes caused by lesions in the central nervous system (as distinct from psychogenic or hysterical paralyzes) fall into two categories, the flaccid and the spastic types.

¹⁶ Peterson, Frederick, in *American Textbook of Diseases of Children* (2d edition). Philadelphia: W. B. Saunders Company, 1938, p. 650.

¹⁷ McIntire, *op. cit.*, *Journal of Psycho-Asthenics*, 1938, 2:44-50.

¹⁸ McIntire, "The Incidence of Feeble-Mindedness in the Cerebral Palsied," April, 1946.

FLACCID PARALYSIS. In the flaccid type, caused by a spinal cord lesion,¹⁹ the muscles below the seat of the cord injury are soft, flabby, and lacking in tonus. The general picture is one of lack of muscle strength (atonicity or hypotonicity). The muscular weakness is apparent in the clumsy, shambling gait, the sagging knees, the affected leg being dragged along in a limp condition. When both legs are involved, the tendency is to walk on the heels rather than on the toes. The reflexes are abolished or diminished in the affected muscle because of the break in the reflex arc. Wasting (atrophy) is a prominent feature in these muscles because they are dependent for their nutrition on the anterior horn cells of the lower motor neurones. The nutritive processes are interfered with in the parts below the lesion. The reactions to faradism (an alternating electrical current from an induction coil) and galvanism (a direct, uninterrupted current from chemical action) are often those of degeneration, whereas no electrical changes occur in the spastic type. Irregular contractions and deformities may develop because of the unopposed action of the nonparalyzed muscles.

SPASTIC PARALYSIS. *Lesions in the upper and lower motor neurone tracts.* Traditionally the paralysis caused by lesions in the upper neurone tracts (in the brain) have been described as spastic (characterized by stiff, contracted muscles and exaggerated reflexes), whereas those produced by lesions in the lower neurone tracts (which extend from the anterior column of the spinal cord to the end plate in the muscles) have been classed as flaccid (characterized by relaxed muscles and diminished reflexes). Although these pure forms may

¹⁹ Injuries to almost any peripheral nerve (outside of the brain and cord) from mechanical wounds, bone and joint lesions, or other causes may also cause localized palsies or related bone-muscle disabilities. Muscular dystrophies also exist without involvement of the nervous system, characterized in different stages by wasting (atrophy), or enlargement (hypertrophy), or both.

Deaver, George C., *Evaluation of Disability and Rehabilitation Procedures of Patients with Spinal Cord Lesions*. New York: Institute for the Crippled and Disabled, 1947.

exist, spasticity is only one characteristic of the complicated symptom-complex technically called cerebral palsy and popularly, but somewhat erroneously, referred to as spastic paralysis. The persons afflicted with cerebral palsy have likewise often been called spastics for short. When the abbreviated term "spastic" is used in the generic sense, it should be clearly recognized that it is a misnomer and includes conditions not characterized by spasticity.

According to one of the New York reports, the true spastic represents about 40 per cent of cerebral palsy cases, 40 per cent are athetoid, and 20 per cent ataxic.²⁰ The 500 New Jersey cases were classified as follows: spasticity, 44.8 per cent; athetosis, 33.0 per cent; tremor, 7 per cent; rigidity, 2.6 per cent; primary incoordination, 4.6 per cent; combination of spasticity and athetosis, 2.6 per cent; all other conditions, 5.4 per cent.²¹ There seems to be an overlap here between spasticity and rigidity; curiously no mention of ataxia occurs, unless it is included in "primary incoordination," and only a small ratio, 2.6 per cent, are listed as having the combined form, in this case only spasticity and athetosis.

In contrast with flaccid paralyses, no muscular wasting occurs in the spastic type except from disuse of the muscles.

Characteristics of cerebral palsy

The brain lesions in cerebral palsy are usually diffuse rather than sharply localized—hence the multiplicity of symptoms—and are static in nature rather than progressive. The symptoms are so multitudinous that W. M. Phelps requires six categories in his grouping of the motor abnormalities, namely:

(1) Tremor, consisting of slow or fast, large or small, automatic rhythmical movements, which follow a consistent pattern in the same antagonistic muscle group, over which the patient has little or no control.

²⁰ *Orthopedically Handicapped Children*, pp. 81 f.

²¹ McIntire, "The Incidence of Feeble-Mindedness in the Cerebral Palsied," April, 1946, p. 492.

(2) Flaccidity, sometimes present as a component of spasticity.

(3) Muscle rigidity or loss of elasticity, a condition of constant stiffness or resistance to movement, caused by diffuse cerebral hemorrhages.

(4) True spasticity, a condition of inconstant stiffness produced by muscle stimulation—the “stretch reflex,” from the action of the antagonistic muscle.

(5) Athetosis, or athetoid movements, characteristic of the “athetoids.”

(6) Ataxia, or ataxic movements, characteristic of the “ataxics.”

The picture is further complicated by the presence in some cases of auditory defects or various kinds of visual defects and a variety of speech impediments. Fifty-four and six-tenths per cent of the 500 New Jersey cases were listed as speech defectives. The three chief motor disorders, spasticity, athetosis, and ataxia, will be described briefly. The latter two conditions may be superimposed upon the muscular spasticity or they may occur independently of it.

Spasticity in cerebral palsy. Spastic paralysis may follow lesions in the motor cortex, in the pyramidal tract (a thick bundle of motor axones from the cortex to the ventral or anterior part of the spinal cord), and in certain organs (basal ganglia) at the base of the brain (the striate body, or corpus striatum). The cerebral motor neurones involved control voluntary movements. Injury to motor cortical areas interferes with the voluntary control of the muscles connected with the given areas. The removal of the cortical inhibition also results in the exaggeration of the reflexes dependent upon the normal functioning of the injured neurones. Loss of voluntary muscle control results, not only from impairment of the cortical motor neurones, but from muscular hypertonicity, irregular automatic movements, exaggerated reflexes, and the simultaneous contraction of antagonistic and reciprocal muscle groups. The muscles in cerebral palsy are often highly

irritable and contract on the slightest stimulation. The spasticity is attributed largely to the excessive contractility produced by the action of the antagonist ("stretch reflex"), which leaves the reciprocal muscle in a state of contraction, instead of relaxation, as long as the stimulation continues. The effort of the patient to execute a voluntary movement is often frustrated by the sudden, automatic contraction of the opposing muscle (stretch reflex). The lesion may produce a reduction or abolition of movement, not from loss of muscle power (paralysis), but from muscle block or contraction. The net result is that the patient cannot perform any movements. When a muscle is placed on tension it contracts and shortens. The continued hypertonicity of the muscles eventually gives rise to contractions and deformities affecting the various parts, such as the hands, arms, legs, and feet. The fingers of the hand may become flexed (see Figs. 21, 23, and 24), the face contorted, and the heel elevated (talipes equinus). In consequence, the patient walks stiffly on the toes. Toe-walking is characteristic of paraplegics. In paraplegia the spasm of the adductor muscle causes the legs to cross over each other in walking (see Fig. 23). This is referred to as crossed-leg propulsion or scissors gait. In hemiplegia the affected stiff leg is dragged along. When the writing hand is involved, the child writes, if at all, in a jerky, angular, unintelligible scribble.

The uncontrollable automatic movements of the affected muscles include tremors, jerks, and spasms, particularly when the corresponding muscles are moved. Effort often results in a magnification of the irregular, incoordinate, and spasmodic movements.

Athetosis. Athetosis, characterized by recurrent, irregular, involuntary movements, results from extrapyramidal lesions in the basal ganglia, especially in the lenticular nucleus of the striate bodies at the bottom of the cerebral hemispheres, and perhaps also in the precentral gyrus. The athetoid movements consist of slow, writhing, twisting, irregular or pattern-

less involuntary movements of the paralyzed parts, such as the shoulders, face, feet, and especially the hands and arms. The contractions may proceed progressively, for example, from the shoulder to the arm, elbow, wrist, and fingers. They are slower and more persistent than choreic movements. When the muscles of the throat, tongue, and diaphragm are seriously involved, as is frequently the case, speech is unintelligible or impossible and the patient may drool and give the impression of being feeble-minded. Thus an adult inmate in an institution for the feeble-minded with severe involvement of the facial muscles, shoulders, and arms, whose speech was almost unintelligible, was given a Binet I. Q. rating in the thirties and had been classified as an imbecile although she was able to read the Bible and could quote numerous Biblical passages. She may have been of average intelligence. Athetoids are usually intelligent, friendly, and unworried. The athetoid movements may pervert voluntary movements into purposeless automatisms. When the legs are involved, the gait becomes very unsteady. Emotional disturbances or voluntary effort exaggerates the condition, and relaxation lessens it. The effort to control the vagrant movements may create tensions that stimulate spasticity. The movements disappear during sleep. Athetosis, found both in paraplegics and hemiplegics, usually does not develop until the second or third year and may be preceded by a period of flaccidity.

Ataxia. Ataxia is characterized by a lack of coordinated action between opposing muscles and a disturbance of balance and posture. It is manifested in awkward movements, diminished reflexes, slurring speech, and the rolling, swaying, staggering gait and station typical of a drunken person. The child is unable to synergize his muscles and coordinate his movements. He cannot execute the movements he wants to make. The condition, however, is not complicated by tension or spasticity. The brain area involved is usually the cerebellum (concerned with the coordination of movements and the maintenance of balance) or cerebellar tract. The

eighth cranial nerve, or the vestibular portion, is sometimes affected. This nerve is concerned with hearing and equilibration.

CLASSIFICATION OF PARALYSES ACCORDING TO PATHOGENIC TYPE

According to the circumstances of the origin, muscular palsies may be subdivided into two groups, the antenatal and natal (it is often difficult to draw a sharp line between these two) and the postnatal. The antenatal-natal group is referred to by a variety of terms, such as cerebral birth injuries, infantile cerebral palsy, infantile cerebral spastic paralysis, or Little's disease (described by William Little in 1862). The latter term is applied by some writers to the whole group of infantile palsy cases, by others to spastic diplegics, and by others to cerebral paralytics with spasticity and mental deficiency. Frequently the term infantile cerebral palsy is applied to all the antenatal, natal, and early postnatal cases.

CAUSES OF THE ANTENATAL TYPE. The most important prenatal factors are brain defects or developmental deviations owing to defective germ plasm (the hereditary elements seem to be particularly prominent in athetosis and diplegia), to abnormal uterine conditions, or to infection of the fetal brain from micro-organisms in the mother's body (such as syphilis, meningitis, or the virus of encephalitis or German measles). Antenatal brain destruction may also be produced by severe hemorrhages caused by antibodies in the mother's blood due to blood incompatibility (when the mother is Rh negative and the child is Rh positive).²² Also, the twisting of the cord around the neck through the turning of the fetus might interfere with the venous return and the oxygenation of the fetus and thus produce neural necrosis (decay) from anoxia of the brain.

²² It is thought that the Rh factor may account for from 8 to 10 per cent of cerebrally palsied children. Perlstein, Meyer A., *The Crippled Child Bulletin*, 1949, II, 8:3.

NATAL CAUSES. The most important natal factors are lack of oxygenation (asphyxia or anoxia) and intracranial hemorrhages. Interference with breathing may be caused by prolonged, difficult labor (which occurs more frequently in the firstborn); breech presentations (legs and buttocks first), which prolong the labor; the use of morphine by the mother as a pain killer ("twilight sleep"), which depresses the breathing center in the fetal brain; and the aspiration (inhalation) of mucous, if the child breathes before he is born, which may obstruct the breathing passages. Hemorrhages may occur in the brain membranes (most seriously in the meninges), in the venous sinuses, and in the brain tissue (intracranial hemorrhages). They may be produced by lack of oxygenation (asphyxia); by excessive blood pressure or venous congestion (which may cause the bursting of capillaries); by precipitate labor, which does not permit proper molding of the head and which may cause lacerations of the meninges from the overriding of the skull bones at the sutures (expansion cracks); by Caesarean delivery, which may not allow time for the process of decompression necessitated by the difference between the uterine and atmospheric pressure; by premature labor (immature blood vessels are very fragile); by forceps injuries (accounting for 5 to 10 per cent); and by lack of vitamin K in the mother's blood before the child is born (without this element the child's blood will not clot).

Molding of the head during the process of birth ordinarily occurs without injury to the brain or the cranium, because the brain is protected by the elastic dura (the outer membrane) and the infant's skull consists of plates of elastic cartilage which are easily compressible and pliable. Excessive molding or distortion, however, from an abnormal presentation or a contracted pelvis, may result in overriding of the bones, change in the balance of the intracranial pressure, laceration of the dura, veins, and venous sinuses, and more or less extensive hemorrhages. Although slight, punctate hemorrhages apparently produce no neurological defects, extensive

ones may result in serious consequences, such as scar formations, adhesions, cell destruction, mental defects, and/or spastic paralysis.²³

Cord injuries may be produced by excessive traction ("obstetrical paralysis"), affecting the upper or lower part of the arm or both, due to injury to the brachial plexus (the network of nerves of the neck and axilla).

The palsies produced by birth traumas include monoplegias, hemiplegias, and asymmetrical paralyses, but not diplegia. Evidences of birth injuries are not found in diplegia, which is characterized by symmetrical spastic paralysis, although the palsy is more marked in the lower than in the upper limbs. Diplegia is usually attributed to neuronc arrest, disintegration, or degeneration of cacogenic origin.

The most serious cases of cerebral palsy are those that result from extensive congenital brain deformities.

The effect of brain injuries on the life expectancy and behavior of the individual varies enormously with the location, extent, and severity of the brain damage. If the lesion is very severe, the child may be stillborn or he may succumb a few days after birth. In the case of a slight lesion (and occasionally in the case of serious ones) no symptoms of any kind may be apparent at birth and the attending physician may report a normal birth. Thus Roberts was able to observe clinical signs of birth injury in only 14 of 48 survivors. William Sharpe reports that scarcely any of his 45 cases could have been diagnosed without a lumbar puncture.²⁴ Penrose believes that "almost every child is intracranially birth injured to some degree even if this degree is very slight."²⁵ Some physicians are of the opinion that as high as 10 per cent of births suffer damage sufficient to produce blood in the cere-

²³ Martz, Eugene, "Recent Trends in the Problem of Cerebral Birth Lesions," *Journal of Psycho-Asthenics*, 1933, 38:311-331.

²⁴ Quoted from Doll, Edgar A., Phelps, Winthrop M., and Melcher, Ruth T., *Mental Deficiency Due to Birth Injuries*. New York: The Macmillan Company, 1932, p. 26.

²⁵ Penrose, *Mental Defect*, p. 111.

brospinal fluid.²⁶ Apparently a large number of children suffer slight birth hemorrhages which are absorbed without leaving any permanent residuals. Doubtless some children slowly recover from birth trauma without giving any evidence of any injury, although some writers believe that some difficult behavior disorders in children for which no explanation can be found may be residuals of unrecognized or subclinical brain lesions.

The fact, however, that no symptoms are apparent at birth is no guarantee that symptoms will not develop later. In point of fact, many symptoms only become gradually apparent, perhaps after a year or two, and so may be attributed to some other cause. The delay in the appearance of the symptoms may be due to the undeveloped condition of the neonatal cerebrum, or, more probably, to the slow progression of the degenerative changes produced by brain inflammations or hemorrhages. The latter may produce areas of sclerosis, increase in glia cells, atrophy, softening, scars, cysts, adhesions, thickenings, cavities (porencephaly), convulsions, and hydrocephaly (from a blood clot at the base of the brain that obstructs the circulation of the cerebrospinal fluid). All of these secondary residuals may produce further degenerative changes. Damaged nerve tissue, unlike skin or muscle tissues, is irreplaceable and unregenerative.

SUSPICIOUS SIGNS OF BIRTH INJURIES. Many symptoms already enumerated constitute suspicious signs of birth injuries, especially the more severe ones, such as muscular twitchings (which commonly point to cerebral lesions), athetoid movements, growing muscular rigidity, paralyses, convulsions, pallid asphyxia, shallow breathing, nursing difficulties, and torpor. Confirmatory evidence should be sought from the examination of the cerebrospinal fluid for the presence of blood and for indications of intracranial pressure, and from a neurological examination. Hydrocephaly is an important sign when accompanied by other evidences of brain trauma.

²⁶ Doll, Phelps, and Melcher, *op. cit.*, p. 36

Note should be made of the development of speech impediments and disturbances that are indicative of paralysis of the musculature of the face and vocal organs. In mild cases the symptoms may not arouse suspicion until the child is several years old. He may be very slow in learning to stand, sit, or walk, and may manifest increasing disturbances in gait and posture and growing muscular stiffness.

PERSONALITY AND BEHAVIOR DISTURBANCES OF THE BIRTH INJURED. Although many of the birth injured, especially those of normal mentality, are well-behaved, stable, agreeable, trustworthy, and industrious, others (especially the mentally deficient and the seriously mentally retarded) are impulsive, emotionally unstable, emotionally immature or infantile, distractible, incapable of prolonged sustained attention, irritable, quarrelsome, unamenable to discipline, and possibly destructive, mischievous, and delinquently inclined. Among 5,000 behavior disorder children in the Institute of Juvenile Research in Chicago, 146, ages one to nineteen, were diagnosed as having "infantile cerebral palsy," and 79 as birth-injury cases without palsy. Sixty-six per cent of the former and 95 per cent of the latter showed behavior difficulties and mental retardation. Both groups displayed somewhat similar conduct disorders, such as violence, lying, stealing, and truancy.²⁷ Some authorities believe that unprovoked, apparently inexplicable, and intractable behavior disorders in children may be caused by birth injuries that were too slight to be recognized or that left no permanent neurological sequelae. Doubtless many personality and behavior deviations in crippled children are caused less by the cerebral injuries they sustain than by the effects of the discrimination, ostracism, ridicule, or the social slights to which they are sometimes subjected by the non-crippled population and which affect them unfortunately.

²⁷ Schroeder, Paul L., "Behavior Disorders in Children Associated with the Results of Birth Trauma," *Journal of the American Medical Association*, January 12, 1929, pp. 100-104.

Estimated intelligence of the cerebrally palsied

The intelligence of children with cerebral palsy is often underrated, especially when the avenues of manual and oral expression are blocked by paralysis of the hands and of the facial and vocal musculature. Among "spastics" encountered by the writer during his first five years of clinical practice were two boys in a special school for mental deficient in St. Louis in the fall of 1914 who had been grossly misdiagnosed. After observing these boys for several months, it became increasingly apparent that they were not mentally deficient at all, although their speech and writing were almost unintelligible and they were able to do only a few of the Binet and the performance tests. Their writing consisted largely of illegible, jerky scrawls, although it improved slowly under the exercises provided for the improvement of manual coordination. When asked why these boys had been assigned to a school for the mentally deficient, the confident reply was: "Anybody could see that they were mental defectives." The fact is, of course, that the diagnosis of the intelligence of many spastics is extraordinarily difficult, and not a simple process of which any amateur is capable. They had been assigned before psychological tests were administered to special class candidates in St. Louis prior to the establishment of the psychoeducational clinic in September, 1914. The mother of one of the boys was prevailed upon to provide a typewriter for each boy to supply them with means of legible communication. With the skill that they soon acquired in hitting the right keys it soon became apparent that these boys possessed far more knowledge and understanding than had been suspected and they were transferred for restoration to grade in an "ungraded" class upon its organization in an elementary school near-by. On my return to the city in 1922 I learned that one boy had been withdrawn from school after finishing the eighth grade, while the other was doing satisfactory work in college.

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Carlson, Earl R., *Born That Way*. New York: John Day Co., Inc., 1941 (a distinguished orthopedist specializing in cerebral palsy, who is subject to spasticity and athetosis).

Hathaway, Katherine, *The Little Locksmith*. New York: Coward-McCann, Inc., 1943; and *The Journals and Letters of the Little Locksmith*. New York: Coward-McCann, Inc., 1946 (both posthumous publications).

Hoopes, Grace G., *Out of the Running*. Springfield, Ill.: Charles C. Thomas, 1938 (birth injury).

Hoskins, Frances H., "The Broken String, An Autobiography," *Training School Bulletin*, May, 1939, June, 1939, 67-75, and October, 1939, 119-123 (a spastic athetoid, who is a writer of poetry).

Children with cerebral palsy vary in intellectual ability from idiocy to supernormality, depending mainly, it seems, upon the extent of the damage to the frontal and parietal cerebral areas related to the higher and lower association processes. Apparently a fair degree of correlation exists between the extensiveness of the brain damage and intelligence as revealed by the extensiveness of the motor involvement (the paraplegics and diplegics rate lower mentally than the monoplegics and the hemiplegics). The spastics, on the average, rate lower than the athetoids and the ataxics. The correlation, of course, may be based on the amount of associated destruction in the "silent" (association) cortical areas. Nevertheless, the degree of the paralysis is in itself an uncertain guide, because too many exceptions to the rule occur. Neither Tredgold nor Shrubsall and Williams found any direct connection between the amount of the paralysis and the

degree of mental impairment.²⁸ A thorough investigation at Vineland of 12 cases revealed "no relation . . . between the severity of the early motor handicap and the later degree of brightness."²⁹ Moreover, they tended to improve mentally as they grew older. Birth-injury cases tend to rate higher intellectually than the antenatal cases caused by developmental perversion or inflammatory diseases of the cortex.

Intelligence as determined by clinical intelligence tests. Fernald and Arlitt found a mean Stanford-Binet I. Q. of 69.1 for 27 birth palsy children in the Cincinnati School for Crippled Children.³⁰ Smith classified 50 cases of "cerebral accidents of childhood," almost 50 per cent of whom were tested during the first three years of life, as follows: normal, 22 per cent; morons, 16 per cent; imbeciles, 40 per cent; and idiots, 22 per cent.³¹

According to Schroeder, the Stanford-Binet I. Q.'s for 146 children with "infantile cerebral palsy" fell in the lower I. Q. ranges as compared with 5,000 behavior disorder children in the Institute of Juvenile Research in Chicago (no averages are given). Seventy-nine "birth-injured" children without palsy had their "greatest frequency at a slightly higher level," and the I. Q. curve for "101 cases with a history of long, difficult labor . . . fell almost directly on that for the birth-injured group."³²

For ten spastics in the Children's Orthopedic Hospital in Seattle, Washington, the Child Study Laboratory of the Seattle Public Schools found a mean I. Q. of 69, as reported by

²⁸ Tredgold, *Mental Deficiency*, pp. 254 f. Shrubsall, Frank G., and Williams, Alfred C., *Mental Deficiency Practice*. London: The University of London Press, 1932, p. 58.

²⁹ Doll, Phelps, and Melcher, *Mental Deficiency Due to Birth Injuries*, p. 249.

³⁰ Fernald, Mabel R., and Arlitt, Ada H., "A Psychological Study of a Group of Crippled Children," *School and Society*, 1925, 449-452.

³¹ Smith, Groves B., "Cerebral Accidents of Childhood and Their Relation to Mental Deficiency," *Proceedings of the American Association for the Study of the Feeble-Minded*, June, 1926, 77-98.

³² Schroeder, "Behavior Disorders Associated with Birth Trauma," p. 102.

Lee.³³ For 20 consecutive cases most recently examined in the author's psychoeducational clinics in Delaware the median Binet I. Q. (Form L) is 65.1 with a range of from 32 to 107 I. Q., a sharp contrast with the corresponding figures for the infantile paralysis cases. Two had I. Q.'s in the thirties, three in the forties, and five in the fifties.

The Stanford-Binet I. Q.'s for 11 of the 12 Vineland cerebrally palsied who could be tested varied from 31 to 94, with a mean of 66.4.³⁴ The three highest I. Q.'s were 90, 91, and 94. The mean for those less than fifteen years of age was 51, and for those fifteen and over, 79, showing a tendency of the intelligence level of birth-injury cases to improve with age, although part of the improvement might be attributable to using a divisor of 14 for those over fourteen years of age.

Of 143 New Jersey noninstitutional cerebral palsy cases, ages 1.8 to 23.8 (median 10.7) tested with the Binet and other tests, 26 per cent were classified as defectives, 13 per cent as dull normal, 13 per cent as low average, 29 per cent as average, 12 per cent as high average, and 7 per cent as superior. Of the defectives 29 per cent were classed as borderline, 27 per cent as morons, 22 per cent as imbeciles, and 21 per cent as idiots. Only 16 per cent of the birth-injury cases were classified as feeble-minded compared with 56 per cent of the congenital intracranial cases. A re-examination of 26 and a prolonged observational study of 22 others did not change the diagnosis. Sixty-three per cent represented birth injuries, 19 per cent were congenital intracranial cases, 7 per cent represented a combination of the two, 4 per cent were encephalitic cases, and 7 per cent were attributed to other causes.³⁵ The more recent tabulation of the 500 New

³³ Lee, Mary V., "The Children's Hospital: A Survey of the Intelligence of Crippled Children," *Journal of Educational Research*, 1931, 164-167.

³⁴ Doll, Phelps, and Melcher, *Mental Deficiency Due to Birth Injuries*, pp. 84 f., 108 f. The book contains a detailed analysis of the performance on each Binet test and on various other tests.

³⁵ McIntire, "Incidence of Feeble-Mindedness in the Cerebral Palsied," *Journal of Psycho-Asthenics*, 44 f.

Jersey cases, based on various psychological tests, the retesting of 87, and other data, is as follows: superior intelligence, 4 per cent; high average, 8.8 per cent; average, 25.0 per cent; low average, 7.6 per cent; dull normal, 11 per cent; borderline, 5.8 per cent; feeble-minded, 27.6 per cent; and undetermined, 10.2 per cent.³⁶

Retabulation of the data for 173 hemiplegics and 114 quadriplegics showed that mental deficiency occurred more frequently among the right-side than among the left-side hemiplegics and among the quadriplegics with greater right-side than left-side paralysis. The incidence of mental deficiency was almost twice as great among the right- as among the left-side hemiplegics and almost two and one-half times as great among the quadriplegics in whom the paralysis was more pronounced on the right side than on the left side.³⁷

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Meyer, Edith, and Simmel, Marianne, "The Psychological Appraisal of Children with Neurological Defects," *Journal of Abnormal and Social Psychology*, April, 1947, 193-205.

Strauss, Alfred A., and Lehtinen, Laura E., *Psychopathology and Education of the Brain-Injured Child*, pp. 98-105.

Obviously, many cerebral spastics constitute special educational problems from the point of view not only of muscular

³⁶ *Ibid.*, *American Journal of Mental Deficiency*, p. 491.

³⁷ McIntire, J. T., "A Study of the Distribution of Physical Handicap and Mental Diagnosis in Cerebral Palsied Children," *American Journal of Mental Deficiency*, April, 1947, 624-662.

re-education, but also of the adjustment of the educational processes and school régime to individual requirements in the classes in which they may be found. According to Doll and Larsen, almost 10 per cent of inmates in institutions for the feeble-minded are cases of cerebral palsy.³⁸

The percentage of cerebral palsy cases in special orthopedic public schools shows a wide variation, possibly because of varying diagnostic standards: Los Angeles, 8 per cent; Newark, N. J., 10 per cent; Chicago (Christopher School), 18.5 per cent;³⁹ Detroit (Oakman School), 21 per cent;⁴⁰ orthopedic classes in Ohio in 1943, 23.6 per cent.⁴¹ The corresponding percentages for infantile paralysis cases, given in the same order, are: 25, 82, 38.2, and 15.4. Fifteen per cent of all crippled children in New York City are reported to be cerebral palsy cases, but probably many of these children were not in special classes.⁴² The number of cerebrally palsied mental deficient in public school classes for the mentally deficient is not available.

Phelps maintains that the ratio of cerebral palsy births is the same every year in all parts of the country, namely, 7 for each 100,000 births. Of the six who survive beyond the fifth year, two are definitely feeble-minded; one is a custodial case, muscularly handicapped beyond rehabilitation, but mentally normal and in need of education; two are moderately handicapped and can be greatly improved, sometimes to the point of complete restoration, and can in due course attend boarding or regular schools; the sixth one (perhaps a spastic hemiplegic) is so mildly affected as to require only some surgical

³⁸ Doll, E. A., "Birth Lesion as a Category of Mental Deficiency," *American Journal of Orthopsychiatry*, January, 1933, 1-13.

³⁹ Heck, Arch. O., *The Education of Exceptional Children*. New York: McGraw-Hill Book Company, Inc., 1940, p. 144.

⁴⁰ According to John J. Lee, as reported by Baker, H. J., *Introduction to Exceptional Children*, p. 152.

⁴¹ Lovinger, Della G., and Nichols, Edith C., *The Cerebral Palsied Child Goes to School*. Columbus, Ohio: Ohio Society for Crippled Children, 1946, p. 5.

⁴² *Orthopedically Handicapped Children*, p. 77.

intervention, or braces, or simple exercises. Phelps estimates that there are 56,000 mentally normal cerebral palsy cases in the United States capable of physical, economic, and social rehabilitation.⁴³

*Treatment and training of children
with cerebral palsy*

No process has been discovered for developing new neural tissue that has been completely destroyed.⁴⁴ All that can be done is to develop the potentialities that may exist in sound adjacent or related cerebral areas and in the musculature.

Muscle re-education. The main reliance for overcoming the spasticity, contractions, incoordination, and athetosis is a comprehensive system of muscle re-education carefully prescribed by a competent examiner to meet the needs of each child and the conditions found in the different body parts—shoulders, hips, legs, arms, hands, face, tongue, etc.—supplemented by certain preliminary operative procedures to reduce the muscle spasm, correct deformities, or equalize the strength of opposing muscles, and by the use of splints, casts, and braces. The results of operative procedures, such as tendon lengthening, transplantation, or tenotomy or muscle transplantation, have often proved disappointing, partly because of undesirable physiological changes. The first objective in the physiotherapeutic approach is to obtain muscular relaxation by placing the child in a supine position in a quiet room, by diverting attention from the spastic member by appropriate psychotherapeutic techniques, and by using muscle-relaxing medicaments, such as prostigmine,⁴⁵ curare, or myanesin.

⁴³ Phelps, W. M., "Recent Significant Trends in the Care of Cerebral Palsy," *Southern Medical Journal*, February, 1946, 132-138.

⁴⁴ A method of hypodermic extraction of blood from blood clots in the brain before scar tissue has been formed, permitting normal brain development in 70 per cent of the cases, has been described. *Newsweek*, July 22, 1946, 59.

⁴⁵ Prostigmine bromide in small tablets lessens spasticity and, given concomitantly, has proved to be a valuable adjunct to re-education and physiotherapy: Jepson, P., "The Use of Prostigmine in the Management of Infantile Cerebral Paralysis," *Journal of Pediatrics*, January, 1946, 65-68.

The treatment begins with slow passive movements of small range with a less severely affected member. The movements should involve complete reciprocation—that is, each pair of muscles should be moved equally far. The immersion of the body parts in warm water, which lessens the tonicity and body weight, is of value, particularly in connection with active movements. Muscle-stretching exercises and the use of weights are contraindicated for spastic muscles. The passive movements should lead the way to active, voluntary cooperation on the part of the child, an indispensable element in the whole process of motor re-education. In the case of paraplegics, much emphasis must be placed on body balancing exercises, on maintaining proper posture in properly constructed chairs, and on stepping or walking with the use of parallel bars, "walkers," ladders (for stepping between the rungs), stiles with banisters, and many other devices. The improvement of muscular coordination in walking, talking, and using the hands and fingers and the prevention of the fixing of contractures by developing correct sitting and standing postures are some of the problems faced by the physiotherapist.

The basic controls can best be established by beginning with infant or amphibian patterns of movement, such as swimming and crawling movements, according to Fay.⁴⁶

The kind of muscle treatment to provide will vary with the type of disability. In the case of rigidity, the emphasis is upon relaxation by movements and medication. Ataxia

See the comments by George J. Boines in connection with Fay's paper. The use of prostigmine has been reported to be "uniformly disappointing" with mentally defective cerebral palsy cases: Hall, Robert J., and Benda, Hans, "Prostigmine Treatment of Cerebral Palsy in the Mental Defective," *American Journal of Mental Deficiency*, January, 1947, 378-383. Further reference is made to prostigmine in the treatment of poliomyelitis.

Clarke, C. Ashley, and Hotson, R. D., "Curare in Oil in the Treatment of Spastic Conditions," *British Medical Journal*, February, 1948, 14:289-291.

⁴⁶ Fay, T., "Problems of Rehabilitation in Patients with Cerebral Palsy," *Delaware State Medical Journal*, March, 1946, 57-60. (See Fay's comments on the "high spinal spastic," caused by injury to the first cervical vertebra and the medulla.)

calls for coordination therapy rather than relaxation. With athetosis and tremor, the primary treatment is relaxation, and movement is secondary. In spasticity the primary emphasis is on the repetition of slow movements to develop motor ability, with simultaneous relaxation as a secondary aid.

Where basic motor controls have been established, physiotherapy should be supplemented by occupational therapy. Many well-known manual and handicraft activities are suitable for the development and improvement of finger coordination, such as threading large beads, placing blocks in recesses, block building, peg board exercises, picture perforation, sticking pins in dots in a pincushion, hitting dots or nailheads on a block of wood, picking up objects, buttoning, unbuttoning, coarse weaving and knitting, rug work, paper cutting, rolling rags into balls, tatting, hemstitching, raffia, basketry, clay work, pottery, jigsaw work, sandpapering, large-scale drawing, sketching, and writing, finger painting, bed craft, shop craft, etc. Such activities should be carefully graded until the power has been developed to tackle more complex handicraft activities or projects. It is of prime importance to begin the training early before undesirable cortico-muscular patterns of response have been acquired and have become fixed. It is important to supply activities with a purpose in terms of the child's own purposing. The muscle re-education program should be made meaningful and dynamic by being integrated with the total program of rehabilitation. Through persistent practice some spastic or athetoid subjects who were unable to hold a darning needle or pen at the onset have become excellent at weaving, macramé, or drawing. But success comes sometimes only at the price of persistent practice for months and years, which, however, must never be carried beyond the point of fatigue.

The result of three investigations may be cited: the first based on a group of 18 institutional mentally deficient birth palsy persons with an age range of from eight to fifty and a Stanford-Binet I. Q. range from 20 to 62 (ten cerebral di-

plegics, five paraplegics, and three hemiplegics),⁴⁷ the second based on 46 mentally deficient cases in the Newark, New Jersey, State School with an I. Q. range of from 29 to 71;⁴⁸ and the third based on a group of 19 cerebrally palsied boys (less one who continued for only two weeks) of average or above average intelligence ranging from five to fifteen years of age (subject to spasticity, athetosis, and "primary incoordination").⁴⁹

Of the first mentally deficient group, who were the recipients of a program of muscle re-education, literary instruction (reading, spelling, and arithmetic), and occupational work for a period of from three to twelve months, all except one increased their Stanford-Binet scores by varying amounts. Six showed marked physical improvement, three moderate improvement, and nine no appreciable improvement. Although none was completely rehabilitated physically or economically, it was held that the mental and social gains were sufficient to justify the training program. The second group of mental deficient was given five years of formal and informal muscle exercises, massage, relaxation treatment, and occupational training in periods of from 30 to 60 minutes, and also academic and muscle instruction and speech training. Some received additional orthopedic treatment, such as tenotomies and the wearing of casts. Thirty-seven and five-tenths per cent improved in walking, talking, using their hands, dressing, feeding themselves, and so on. Even some low grades learned to walk and talk understandably. Improvement depended more on cooperativeness and effort than on the degree of intelligence as determined by tests. Most were rated as mentally normal after their interest and enthusi-

⁴⁷ Martz, Eugene W., and Irvine, Helen N., "The Results of Physical and Mental Training on Mentally Deficient, Birth Lesion Children," *Journal of Juvenile Research*, 1934, 42-51.

⁴⁸ Sirken, Jacob, "Treatment of Cerebral Palsy," *American Journal of Mental Deficiency*, April, 1941, 544-547. See also this author's earlier article in *Journal of Psycho-Asthenics*, 1939, No. 2, 107-113.

⁴⁹ McIntire, J. T., "Cerebral Palsy Treatment Experiment," *The Crippled Child*, December, 1942, 94-96, 111.

asm had been aroused, although they had been classed as idiots prior to the program of rehabilitation.

It was estimated that the majority of the mentally normal group that received a program of systematic relaxation and specific muscle re-education in the Babbit Hospital in the Training School at Vineland would, as adults, "be independent, a good per cent of the remaining cases will prove to be partially self-maintaining, and only a few will be dependent in the ordinary sense." Improvement in social maturity or competence was marked in some cases, as measured by the Vineland Social Maturity Scale (which measures advancement in self-help, self-direction, locomotion, occupation, communication, and social relations).⁵⁰ Half a dozen were able to walk without much assistance. Although 70 per cent before admission to the hospital had, because of their physical handicaps, attended special schools or classes or received home instruction, 77 per cent upon discharge were able to attend the regular classes, leaving only 23 per cent in need of special education. Although all had improved in varying degrees, no one was completely rehabilitated.

Academic, occupational, and vocational education. The coordinated or total plan of rehabilitation includes programs of education adjusted to the needs of the individual child as determined, not only by orthopedic and neuromuscular examinations, but by examinations of vision, hearing, speech, intelligence, personality structure, social characteristics, educational potentialities, abilities, and disabilities, and vocational predilections and possibilities. Objective tests so far as feasible should be skilfully employed for clinical analysis of each child's assets and deficits and must be carefully evaluated in the light of the synoptic findings.

The academic and muscle rehabilitation programs should be conducted concomitantly. The educational program should be provided, according to the age, condition, and intelligence of the child, in nurseries, nursing schools, hos-

⁵⁰ Doll, E. A., "Birth Lesion as a Category of Mental Deficiency."

pitals, convalescent homes, camps,⁵¹ rehabilitation centers, residential institutions for the mentally defective or the orthopedically handicapped, special classes for the mentally retarded in orthopedic schools when available, in the regular grades, or in the homes through home teachers or two-way radio instruction.⁵² The latter device, in which Des Moines, Iowa, has pioneered, enables the home-bound child to listen in on activities of his classroom and to participate in the same. In hospitals use is now being made of microfilm books which are projected on the ceiling, where they can be easily read by the bedridden child.⁵³ Those who are mentally normal should be retained in special orthopedic schools only as long as they need the special physiotherapeutic, orthopedic, and educational services afforded by such schools or classes. Whenever the classes are in the regular grade building, when it is practicable the children should be permitted to attend the regular grades in activities in which they can compete without too much of a handicap. Abundant opportunities should be afforded for socializing group or cooperative activities on all levels.

Cerebral palsy cases of idiot and imbecile grade should be given needed care and training in institutions or colonies for the mentally deficient. Those of higher imbecile levels should be admitted on probation to public school special classes for the mentally deficient. Some, who will eventually prove to have been pseudo-imbeciles, will make greater progress under skilled teaching than had been anticipated. Psychological diagnosis must be tentative in the case of those whose powers of language and motor expression are gravely impaired.

Fully 50 per cent of these children require speech therapy.

⁵¹ *A Bibliography on Camping with Handicapped Children*. Chicago: National Society for Crippled Children and Adults, 1948.

⁵² Winterstein, W. A., "School by Telephone," *The Child*, April, 1941, 250-251.

⁵³ Holway, Marian, "The Ceiling Reflects New Hope for the Handicapped," *Modern Hospital*, February, 1948, 79-81.

administered, preferably, by correctionists with special training in speech work with the cerebrally palsied. With some cases in which no organic defects exist in the musculature of the speech organs, the point of approach should be that of mental hygiene rather than that of articulation drills. Often the process of speech development and correction involves a combination of mental hygiene therapy and muscular and articulation exercises.

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The whole school program should be pointed toward making social and economic assets so far as possible of cerebrally palsied children. An important phase of that program is the prevention, mitigation, or cure of the personality maladjustments or handicaps that often develop from the multiphase conflict situations that beset the cerebrally palsied because of their physical limitations. Occupational and vocational training should be provided to meet individual penchants and talents. Suitable vocational training will vary according to capacity, specific ability or disability, and interest, from automatic, repetitive routine work to skilled jobs or to semiprofessional or professional service occupations. It is important always to capitalize on the talents and potentials that remain and can be converted into vocational assets. The departments of vocational rehabilitation can contribute to the vocational objectives by providing opportunities for the cerebrally palsied of "employable age" with "static disabilities" that can be "eliminated or be substantially reduced . . . within a reasonable length of time."

State-wide programs for cerebrally palsied children. Under the federal grants-in-aid made available to the states on February 1, 1936, for medical, surgical, and aftercare services for the physical restoration and social readjustment of crippled children administered by the U. S. Children's Bureau, all the states, Alaska, Hawaii, and the District of Columbia have established state agencies to supervise the program. In 1944 the federal grant amounted to \$3,781,751.77. Presumably children with cerebral palsy are enjoying the benefits of these services in most, if not all, of the states. In many states

ILLUSTRATIVE CASES. The diagnoses and data for the subjects shown in Figs. 21, 22, and 23 are reproduced from one of the author's publications,⁵⁴ and those for the subjects shown in Fig. 24 (which includes one infantile paralysis case) were extracted from the case files of the Elwyn Training School in Pennsylvania.

F. H. (Fig. 21) is an imbecile afflicted with spastic right hemiplegia, or paralysis of one side. Age when photographed 18.2.



FIG. 21. F. H., spastic paralysis of the right side (hemiplegia), age 18.2, Binet age 4.6, I. Q. 27.*



FIG. 22. W. E., spastic paralysis of lower limbs (paraplegia), age 11, Binet age 6.3 at the age of 10.4, I. Q. 59.†

His Binet age a year earlier was 4.6, and his I. Q. 27. Right arm and leg atrophied, right hand contracted, wrist flexed, hearing and vision poor on right side, nose deviates to right, facial paralysis. Illegitimate. Walked and talked at four. Convulsions involving the whole body since that time. Usually good-natured,

⁵⁴ *Education of Handicapped Children*, pp. 340-341.

* Wallin, J. E. W., *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1939.

† *Ibid.*

but inclined to be quick-tempered, irritable, fretful, and stubborn. Cannot wash, dress himself properly, read, or write, but does simple, outdoor industrial work that can be done with one hand, such as sorting wood, and picking up stones and papers.

W. E. (Fig. 22), who has spastic paraplegia, or paralysis of the lower limbs, was eleven years old when photographed. His Binet level was 6.3 at the age of 10.4, giving an I. Q. of 59. Instrumental birth, with asphyxia. Severe fever at six months, feet drawn up. Uttered words when one year old, but did not walk until eight. Spastic gait. Knees semiflexed, cannot be extended. Left talipes equinus (elevation of heel, walking on toes). Does not read or write, but does kindergarten work, knows color and form, can string beads, do simple weaving, simple hand work, lace, button, place pegs in peg board, and play with toys. Good disposition.

Y. G. (Fig. 23) is an idiot afflicted with spastic diplegia or quadriplegia (paralysis of both sides) who has never been able to walk or creep, or sit straight in a chair, or talk. Cannot bend body. He pulls himself along on the floor on his stomach. Low hair line. Drools. Fretful, stubborn, and subject to passionate outbreaks of temper. One sister



FIG. 23. Y. G., spastic paralysis of both sides (diplegia), age 17, Binet age between one and two at the age of 12.*

a spastic diplegic idiot. Photographed at age of seventeen. At age of twelve had a Binet age between one and two. Can recognize toys, distinguish candy from wood and remove wrapping, can make marks with a pencil, and obeys commands so far as he is physically able to do so.

J. A. (Fig. 24, No. 1) recorded as birth injured (did not cry for 36 hours, mouth open), entered the institution at the age of

* *Ibid.*

7.10 with partial paralysis (hemiplegia); walks fairly well; speech affected; reached about fourth grade in reading comprehension and spelling; has assisted in mattress shop for years; very cooperative, always willing to help, but sometimes a nuisance because he overestimates his ability and interferes with others; somewhat egotistical; wants to be in the limelight. Photograph at age thirty-two.



FIG. 24. Various kinds of orthopedic defects: (1) birth injury; (2) birth injury; (3) poliomyelitis; (4) brain cyst; (5) birth injury.*

H. E. (Fig. 24, No. 2), admitted to the School as a birth-injury case at the age of 7-3; legs spastic (paraplegia); scissors gait; hallus vulgus (big toe bent toward other toes); talipes equinus partly corrected; knock knees; internal "squint" (strabismus); nervous blinking; abdomen spastic. Did not walk until three. Stanford-Binet age 11-9 and I. Q. 73 at 35-4; Form L Binet age 12-3, I. Q. 82, at 47-8. Has worked in the shoeshop for years and also assists in the candy store. Plays the accordion. Has excellent vocabulary and "plenty of good sense, but is hampered by his physical condition." Is interested and friendly, but has been under-

* Courtesy of E. Arthur Whitney.

handed, instigating others to run away although he does not indulge in this prank himself. Photograph at the age of fifty.

R. H. (Fig. 24, No. 3), admitted at about the age of fifteen, with complete paralysis of the left leg, partial paralysis of the right leg, both legs atrophied from poliomyelitis, but upper limbs well developed; uses crutches; Stanford-Binet age 10-6, I. Q. 70, at seventeen; Form L age 11-2, I. Q. 74, at twenty-nine; at eighteen still in school in afternoons in woodwork; has done satisfactory work for years in the shoeshop; also assists in candy shop; "keeps other boys straight; intelligent, very popular, everybody's friend, most optimistic disposition, 'father' of the infirmary." About thirty-one at time of photograph.

T. B. (Fig. 24, No. 4), admitted to the school at the age of eleven, with spastic paralysis and atrophy of the right leg, arm, hand, and shoulder (hemiplegia), caused by a small porencephalic cyst in the region of the left lateral ventricle toward the cortex (operated upon at age eleven; a large scar area could not be removed). Walks with slightly spastic gait affecting the right leg. At 19-2 had a Form L Binet age of 6-5, I. Q. 43; at 21-0, a Form L age of 8-0, I. Q. 53. At seventeen had reached about second grade in spelling, but had improved little in arithmetic; at twenty tried all kinds of handwork and was doing easy weaving. Has torn and sewed rags for years. "A nice boy," tries hard, patient, enjoys attention, sensitive, easily hurt by criticism; "quite a teaser." Photograph at the age of 21-8.

J. H. (Fig. 24, No. 5), hemiplegic, admitted at the age of 19-7 and recorded as a "birth-injury" cripple (long difficult labor, requiring artificial respiration, feet born first), with meningitis in infancy. At 19-8 had a Form L Binet of 6-0, I. Q. 40. At that time could count to 1,000 and read small words, was excitable, but affectionate and truthful. At twenty-four, when photograph was taken, was characterized as "quiet, peaceful, and friendly; but blank: accomplishes nothing."

Brain injuries without motor involvement

It is now recognized that the effects of brain injuries of prenatal, natal, or postnatal origin may be reflected in perceptual, conceptual, personality, behavioral, and learning disorders rather than in motor disabilities, and that such children stand in need of highly differentiated diagnostic educational treatment if their disabilities are to be ameliorated or

removed and their energies released into socially acceptable and constructive channels.⁵⁵

These children are ordinarily characterized by high distractibility, undue fixation of attention on irrelevant stimuli or unessential details, abnormal responsiveness to certain stimuli, lack of continuity of effort or perseverance, and sometimes perseveration (repetition of the same responses, such as numbers or tapping). The perceptual disturbances, which may affect the visual, auditory, and kinesthetic senses, may manifest themselves in a confused perception of number relations, letters, and words, in skipping words or reading them out of order, and in perceiving objects as disparate entities rather than as parts of larger unities. On the behavioral side many of these children tend to be hyperactive, disinhibited, explosive, emotionally unstable, noisy, careless, and they sometimes do things at a breakneck speed. They are often excessively meticulous, exacting, formalistic, or pedantic, indecisive, erratic, flighty, given to daydreaming, and fantastic or incoherent in their thinking. With these handicaps, learning is prevented or impeded without the use of special remedial techniques.

Palsied children often show some of the traits of the birth injured without motor involvement, such as distractibility, emotionality, restlessness, and hyperactivity.

The suspicious signs of this type of brain injury correspond in part with those given on pages 221 and 433. But evidences of motor disabilities are lacking except in unusual cases. The brain lesions are usually found in the extrapyramidal system (basal ganglia), especially in the thalamic region,⁵⁶ as shown by disturbed reflexes affecting the eyes (nystagmus and light reactions), the hands and wrists, the big toes, the abdomen on one side, and by athetoid movements. Teach-

⁵⁵ See the elaborate treatment of the subject by Strauss and Lehtinen, *Psychopathology and Education of the Brain-Injured Child*, pp. 86, 106-116.

⁵⁶ The thalamus, one of the largest ganglia in the extrapyramidal system, relays visual, auditory, tactual, kinesthetic, pain, and temperature impulses to the cerebral cortex and is the motor center connected with emotional behavior.

ers, social workers, and psychologists who suspect the presence of the not easily diagnosed non-motor kind of brain injury from the child's abnormal reactions should see to it that the case is referred for a competent neurological diagnosis.

To overcome these and other difficulties Strauss and Lehtinen have devised a comprehensive set of didactic procedures administered in a neutral, nonstimulating environment (with the child facing the wall at close range or isolated behind a screen and with distracting details, such as attractive pictures on the walls and in the books, removed), which is designed to control vacillating attention, to inhibit or limit movements (for instance, by the use of colored borders, which the hand movement must not exceed), to slow down precipitate movements (through placing screws in holes, or the use of a marker or finger in reading), to require orderly procedures, to develop visual discrimination of spatial relations (geometrical forms, puzzles, letter forms, words) and number relations (through exercises with the colored beads on an abacus, and the separation of words by colors), and to develop the perception and integration of wholes (through the reproduction of patterns in red and black marbles on a marble board). Much concrete manipulative work is provided for perceptual training and as an outlet for the motor impulsiveness. Writing is provided not only as a means of communication, but as a means of developing visuomotor perception and learning to read. The exercises provided involve the solution of intellectual problems and the gaining of insights. These and many other external controls and crutches are gradually removed as the handicaps are overcome. Teachers with brain-injured children should familiarize themselves with the detailed remedial techniques outlined by Strauss and Lehtinen for the teaching of reading, arithmetic, and other subjects.⁵⁷

CAUSES OF THE POSTNATAL TYPES. The postnatal forms.

⁵⁷ *Ibid.*, pp. 127-189.

which may be either flaccid or spastic, can be grouped into three main categories:

(1) Those caused by bacterial infections, the most numerous of which are noted below.

(a) Cerebrospinal meningitis, an infection of one of the meninges or brain covers (especially the delicate pia mater which adheres to the cortex). The crippling effects of the pneumococcus variety, formerly always fatal, can now be largely cured through the early use of sulfa drugs, such as sulfamerazene or sulfadiazine, and penicillin. The Simon Flexner serum is effective with some varieties.

(b) Tuberculosis of the bones and joints, especially of the spinal cord, hip joint, and the bones of the limbs, usually secondary to tuberculosis in some other part of the body. Deformities often result from the degenerative process. These cases are less frequent now than formerly, constituting 2.4 per cent of all the crippled children reported by the U. S. Children's Bureau in December, 1944. Streptomycin is now used with good results in treating many types of tuberculosis.

(c) Rheumatic fever (acute articular rheumatism) and arthritis, some varieties of which apparently are caused by a streptococcus infection. These groups, not all microbe-produced, constitute 3.5 per cent of the Children's Bureau 1944 figures.

(d) Osteomyelitis, acute or chronic, a painful inflammation of the bone marrow or of the bone and marrow, especially of the legs, probably caused by infection from different kinds of micro-organisms (such as the staphylococcus). Osteomyelitis comprises 4.6 per cent of the Children's Bureau total. It is now possible to prevent or to cure the disease by the early use of sulfa drugs and penicillin.

(e) Anterior poliomyelitis, discussed in Chapter 18.

(2) Those caused by specific mechanical traumas, such as burns and particularly falls, concussions, or blows that produce fractures or dislocations, or serious injuries to the brain,

spinal cord, or peripheral nerves. Accidents occur chiefly in the homes, playgrounds, and streets (particularly from the automobile traffic). Burns and other injuries account for 8.3 per cent of the Children's Bureau's cases.

(3) Miscellaneous orthopedic conditions that create special education problems, such as flat feet, abnormal spinal curvatures (scoliosis or lordosis), rickets (or rachitis), and progressive muscular dystrophy.

PROGRESSIVE AND PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY. Progressive muscular dystrophy, which is confined almost exclusively to the male sex, is usually described as a primary disease of the muscles associated with a disturbance in muscle metabolism involving the creatine element, of unknown origin except that evidence of heredofamilial incidence has been found in about 60 per cent of the cases. Two or more sibs and other members of the fraternity are sometimes affected. Several forms of the malady have been identified, occurring at different stages from infancy to adulthood.

The most prevalent form in childhood, known as pseudohypertrophic muscular dystrophy, which ordinarily begins between the second and seventh year, is characterized by progressive atrophy and splitting of the muscle fibers and enlargement of the muscles from an accumulation of fat tissue which replaces the atrophied muscle tissue (hence the muscle is referred to as pseudohypertrophic). There follow increasing diminution of the reflexes, muscle weakness, and paralysis, without any impairment of sensitivity. The process of muscle degeneration is frequently accompanied by mental deterioration. Five of Yannet's cases were diagnosed as morons.⁵⁸ The enlargement is particularly noticeable in the calves of the legs, the forearms, and the shoulder girdle. Usually the first symptom is growing weakness of the legs, first one and then the other, and high fatigability. The gait becomes increasingly unsteady, the child stumbles and falls

⁵⁸Yannet, "Diagnostic Classification."

more and more and experiences increasing difficulty in climbing stairs and in getting up from the floor. He rises slowly by pushing his hands against the floor or against the thighs. Eventually little use can be made of the hands because of the growing weakness of the arm muscles. The disease progresses very rapidly in some cases and very slowly in others, sometimes remaining stationary for some years. As a result of the growing paralysis, the child eventually becomes helpless in a wheel chair or in bed until he succumbs, ordinarily from some intercurrent disease, often in early adolescence.

No method of preventing or curing the condition has been discovered. The palliative treatment includes systematic exercises of the extensor muscles, massage, the wearing of supports for weak ankles or a weak back, the operative lengthening of contracted tendons, the use of a diet of high vitamin C and D content, and the administration of various drugs (for instance, glycine) of dubious value.

These children are referred to the psychoeducational clinics in the schools and to mental hygiene clinics because of growing muscle disabilities and failure in studies. Many had a record of satisfactory progress in school prior to the onset of the symptoms. Almost all of those examined in the author's clinics have evidenced various degrees of mental retardation. The Stanford-Binet I. Q.'s (Form L) of the last seven consecutive cases, all boys, varying in ages at the time of the examination from 8.1 to 17.3, are 52, 64, 69, 74, 80, 87, and 93. The boy with an I. Q. of 87 at the age of 9-10 had an I. Q. of 76 at the age of 11-4. From the standpoint of mentality, several of these boys distinctly belonged in special classes for the mentally deficient, where they should be permitted to remain, provided with comfortable seating arrangements (wheel chairs) until they become too much of a burden because of the neuromuscular deterioration. Only one of these boys (I. Q. 80) had attended an opportunity class. His brother, whose psychological record was unavailable, also was afflicted with muscular dystrophy.

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Chapter 18

THE ORTHOPEDICALLY DISABLED: POLIOMYELITIS

*History of infantile paralysis (poliomyelitis
or Heine-Medin disease)*

This acute neurotropic disease, which apparently occurred among ancient Egyptians about 4,000 years ago, is said to have been first recognized as a distinct entity in 1840. The modern world outbreak started in Sweden in 1881 and rapidly spread to Europe and the northeastern part of the United States. The worst outbreaks in the United States were in 1916, with about 29,000 cases and in 1946 with 25,191 cases, and in 1948 with 27,658 cases. Nearly 10,000 cases are reported in the United States during the average year. In 1947, 10,000 cases were reported in England.

The disease occurs in both sporadic and epidemic forms; the epidemic form is more severe, especially in the early part of the outbreak. The disease occurs in all degrees of severity, from a barely recognizable illness or infection which might resemble a cold or attack of influenza (subclinical form) to almost total paralysis of the affected limbs. The so-called abortive form, perhaps the most prevalent one, may consist merely of a fever, vomiting, sore throat, headache, and upset stomach. Fortunately, even in the severest epidemics, only about 1 person in 1,000 and 1 child in 300 under the age of five contracts the disease in a form severe enough to show definite symptoms. This is assumed to be due to the fact that immunity is acquired even from the slight abortive or unrecognizable at-

tacks. One attack almost always confers permanent immunity. Although the disease affects both adults and children, the vast majority of cases are children under fourteen. Of 2,958 cases in Chicago and Detroit between 1939 and 1944, 23.1 per cent were under five, 36.1 per cent were between five and nine, and 23.5 per cent between ten and fourteen. Of those under twenty-five (95 per cent), 58.7 per cent were boys and 41.3 per cent girls. Apparently, the incidence of the disease is increasing among adults. Thus, at the present time, 25 per cent of the victims in cities in the United States are over fifteen as compared with 3.7 per cent in 1916. In Copenhagen in 1944, 53 per cent of the cases were over fifteen. No climatic restrictions exist and there is no racial immunity. The greatest incidence occurs in the late summer and early fall, with merely traces of sporadic cases between December and May. The geographic distribution of the outbreak follows an erratic course from year to year, although the epidemics tend to move in cycles of from four to six years. The fatality rate differs in different epidemics according to the severity of the disease. The mortality rate among 19,029 cases in 1944 was 7.1 per cent as compared with 3.1 per cent for Maryland cases in 1941. Early fatalities are commonly caused by paralysis of the muscles of respiration or circulation from lesions in the respiratory and heart centers in the bulb (medulla oblongata). Of 245 fatal cases in the Minnesota epidemic in 1946, 44 per cent died within a week after the onset, whereas only 17 per cent lived longer than a week and none over two years. New muscles were seldom involved after the first week (E. T. Bell). The fatality rate is greater for adolescents and for adults than for children, and greater for males than for females. The basis of susceptibility to the disease remains undetermined, although Neil N. Litman and James F. Bosona maintain that "growth failure and susceptibility to clinical poliomyelitis are related phenomena."¹

¹ As reported in *Science News*, July 21, 1948.

During epidemics it is important to examine very early the spinal fluid for increase in mononuclear cells and globulin contents of all persons who show suspicious symptoms of infection in order to confirm the diagnosis if possible. Patients should be isolated and proper treatment instituted without delay.² The disease is most contagious during the first 10 days of the infection.

Nature of the disease

The ultramicroscopic organism that produces poliomyelitis in man and monkeys³ has not been identified as being a filtrable virus. It is probable, however, that several varieties of viruses may produce different varieties of polio. The polio viruses are among the tiniest of known organisms—two or three million to an inch—and also the toughest. They have remained active in sterile water for 114 days and in stools or sewage for over six months, although they feed only on a living animal as a “host,” or on a medium containing living tissues. They are immune to ordinary germicides, but can be destroyed by ultra-violet rays, formalin, bichloride of mercury, hydrogen peroxide, and a temperature of over 130 degrees.

The chief portals of entry of the pathogenic organisms are the mouth and upper respiratory tract. They apparently are transmitted from the nose, throat, and bowels of a sick person and, especially, from a carrier to the affected person by means of droplets thrown into the air through the process of exhalation, coughing, or otherwise; or through the ingestion of milk or other foods that have been contaminated; or through the intake of water contaminated by sewage. The unwashed

²For critical commentaries on the spinal fluid tests see Boines, George J., and Pollak, Otto J., “Evaluation of Spinal Fluid Examination on Patients with Poliomyelitis,” *Delaware State Medical Journal*, August, 1946, 171-175.

³In 1908, Karl Landsteiner of Vienna infected monkeys with polio by inoculating them with virus isolated from human beings. In 1939 Charles Armstrong found that the eastern cotton rat could also be infected with certain strains of the virus.

hand is doubtlessly often the medium of transmission through the handling of food, or, in the case of children, through the insertion of the fingers in the mouth.

Investigations in Detroit, New York City, Charleston, South Carolina, and Stockholm, Sweden, show that the polio virus is carried in sewage, principally from hospitals. In 1941 the experiments of John L. Paul and James B. Trask demonstrated that rapid crippling can be produced in animals injected with the virus from flies that have had access to open sewers and privies in epidemic areas.

The virus invades the nervous system directly, having a special affinity for the gray matter in the bulb at the upper end of the cord (bulbar poliomyelitis) and more particularly in the anterior horns of the spinal cord, especially in the lumbar enlargement; hence the term anterior poliomyelitis (*polios*, gray; *myelos*, marrow). As already explained, the bulb contains the nervous centers that control the muscles of the throat, the chest, and the heart.

The incubation period (dating from the entrance of the virus to the appearance of the first symptoms) of from four or five days to ten or twelve days is followed suddenly by the acute stage, lasting from one to four weeks. The acute stage is classified according to the severity of the disease as follows: during the epidemic months one may expect to see a number of patients who give the history of sudden onset, malaise, fever, upper respiratory infection, and possibly nausea and vomiting. Such a patient could easily have been the victim of an abortive type of the disease. However, since we have no method of virus diagnosis, this is classified as an upper respiratory infection. When the symptoms described above are followed by headache, drowsiness, muscle tenderness, hyperesthesia, stiffness of the neck, back, hamstrings, and calf muscles, the picture is one of nonparalytic poliomyelitis. The deep reflexes may be diminished or absent, and muscle twitching (or fasciculation) may be present, which further strengthens the diagnosis. The patient is usually unable to sit up

without leaning backward and supporting himself by placing both hands behind him on the bed. In the spinal paralytic patient, all of the above symptoms are present in addition to definite muscle weakness or paralysis of some muscle groups which comes on suddenly and is usually asymmetrical. The paralysis may involve one or both legs and arms—and many other muscle groups, such as those of the abdomen and trunk. There may be urinary retention if the bladder is involved. Invasion of the bulb is reflected in difficulty of swallowing, speaking, and breathing. Five different kinds of bulbar involvement have been distinguished: (1) the cranial nerve nuclei type, which affects the muscles of the face and throat and produces difficulty in swallowing and possible death from choking; (2) the respiratory center type, which produces loss of control of the breathing rhythm and possible death from anoxia (oxygen deficiency); (3) the circulatory center type, which produces shock effects through interference with the circulation and lowering of the blood pressure; (4) the encephalitic type, which produces oxygen deficiency in the brain and, perhaps as a consequence thereof, confusion, apprehension, and anxiety; and (5) the bulbar cervical type, which affects both the bulb and the adjacent spinal areas, and produces a mixture of the symptoms of the other types and paralysis of the upper torso (especially the chest respiratory muscles).

The return of muscle power often begins spontaneously during the first two weeks. In most cases the palsy is partial; most muscles are weak rather than totally paralyzed.

During the convalescent stage, beginning with the cessation of the muscle tenderness and continuing to the point where spontaneous improvement has largely ceased, about two years under the traditional treatment, the affected muscle will improve in strength, but muscle and tendon atrophy (largely from the destruction of the anterior horn cells) and deformities and limb shortening may also develop. But these untoward effects can be prevented to a large extent by careful muscle stretching, avoidance of excessive fatigue, and by sup-

plying needed muscle training and appropriate physiotherapeutic and hot fomentation treatment. Prostigmine, curare, and myanesin are used to relax muscle spasm.

During the stationary chronic stage, which may continue throughout life, little spontaneous improvement occurs in those who have not completely recovered; but much improvement is possible from proper muscle treatment. The after-effects of serious neuronc inflammation or destruction of the cells (the organism feeds on the nerve cells) remain as permanent scar tissue or gliosis. Nerve tissue that has been completely destroyed cannot be regenerated, but the nerve damage in many cases is only partial. Of 296 Maryland cases 50 per cent were listed as being free of aftereffects, 29 per cent as having slight residuals, and 18 per cent as having marked muscular disabilities. Symptoms that are indicative of a favorable prognosis are slightness of the muscle weakness and its diffuse nature, rapid recovery of muscular strength after the subsidence of the acute infection, and the mildness or abatement of the muscular tenderness. The outlook is discouraging so far as concerns restoration of seriously palsied muscles that have shown little improvement for six months under skilled therapy. The efficacy of the treatment decreases with the lapse of time. Less can be accomplished during the second than during the first six months, and less during the third year than during the second year; but slow progress may even accrue thereafter, particularly if effective physiotherapeutic treatment is continued. The importance of proper treatment is shown by the following figures for 97 cases treated (muscle training and rest) and 60 untreated cases from Vermont: 100 per cent of the former showed improvement as against only 27 per cent of the latter.⁴

Preventive treatment

Little progress has thus far been achieved, in spite of the millions realized annually through the recurrent financial drives conducted by the National Foundation for Infantile

⁴ Jones and Lovett, *Orthopedic Surgery*, p. 431.

Paralysis, toward the realization of the ultimate objective of all efforts in connection with this infection, namely, its prevention by means of vaccines or immunizing agents. Almost all of the numerous preventatives that have been announced from time to time during the last ten or fifteen years have fizzled out. Preventive measures most frequently emphasized, especially during epidemics, necessarily of limited efficacy, are: keep children away from crowds, especially new groups of people (which may contain carriers); don't swim in polluted water or in pools with crowds; wash the hands before eating; keep contaminated objects out of the mouth; screen the house against flies and insects; keep flies off food; destroy garbage and waste; avoid excessive fatigue and body chilling; don't remove tonsils or adenoids prior to or during epidemics; avoid exercise after the first onset of the symptoms and call the doctor at once. The value of draining pools and swamps and dusting infected areas with DDT has not yet been determined. The value of mass destruction of flies and mosquitoes by spraying of DDT by planes and by ground units is now under study by the United States Public Health Service. It is probable that flies may play a part in the transmission of the poliomyelitis virus from infected sewage or open toilets to food or water used by susceptible individuals.

The intelligence of polio cases

In the Cincinnati group studied by Fernald and Arlitt the median Stanford-Binet I. Q. was 83.7 for 62 polio cases as compared with 69.1 for 27 spastics; the corresponding figures for 26 polios and 10 spastics in Seattle tested by the Child Study Laboratory of the public schools are 92 and 69. In a group of 300 crippled children of various kinds in Connecticut, Massachusetts, and New York examined by Mildred Stanton, the mean Form L Binet I. Q. for the polios was 94 as compared with 88 for the entire group.⁵ A. C. Williams's intelligence

⁵ Quoted from Pintner, *et al.*, *Psychology of the Physically Handicapped*, p. 270.

classification of 490 children in schools for cripples in England is as follows:⁶

	<i>Above Average</i>	<i>Average</i>	<i>Below Average</i>	<i>Nearly Defective</i>
Poliomyelitis	18.1	48.7	25.6	7.5
Cerebral Diplegias	0.0	15.1	24.2	60.6

In a later investigation of 98 polios, ages four to sixteen, in a British orthopedic hospital and clinic, Gordon, Roberts, and Griffith found a Binet I. Q. range of from 50 to 149, with a mean of 103.9, as compared with a mean of 98.8 for the total school population of Bath.⁷ The average Binet I. Q.'s (Form L) for nine consecutive polios most recently examined in the writer's Delaware clinic is 107, with a range of from 79 to 122. Only one child had an I. Q. under 100, namely, 79.

These limited surveys seem to show that the typical polio case possesses average or nearly average intelligence. However, a larger ratio of minus than plus deviates exists among polios. An appreciable proportion of these children are subnormal mentally, but the subnormality is primary and not secondary to the infection except in the few cases in which the "silent" cortical areas are involved. Involvement of the cerebral neurones seems to make for irritability, lack of sustained attention, and impaired comprehension of spatial relations.⁸

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⁶ Penrose, *Mental Defect*, p. 116.

⁷ Pintner, *et al.*, *Psychology of the Physically Handicapped*, p. 270.

⁸ Crothers, Bronson, and Meyers, Edith, "The Psychologic and Psychiatric Implications of Poliomyelitis," *Journal of Pediatrics*, March, 1946, 324-326.

Treatment

Acute phase. Treatment is now directed toward the general condition of the patient with intravenous fluids, transfusions, and supportive therapy; hot packs and nursing care, according to the Kenny method; and early mobilization and muscle training. The use of relaxant drugs has been introduced lately for the relaxation of the muscle spasm which is always present in polio. The spasm and shortening of the muscles cause deformities unless the spasm is properly relaxed. In addition to the above care, bulbar cases require special treatment because of the fact that the patient is unable to swallow his own saliva due to paralysis of the palate and pharynx or because of the involvement of the chest or heart muscles. The patient is placed on his side or abdomen with the foot of the bed raised to facilitate the drainage of saliva and mucous. This is also facilitated by suction of the saliva through a tube from the mouth or through an opening in the windpipe (tracheotomy) and by mechanical respiration and oxygen inhalation in some of the bulbar cases. According to A. B. Baker, not over 10 per cent of bulbar cases should succumb under proper treatment, most of these from injury of the blood circulation center for which no effective treatment is at present available. During the 1948 epidemic a new synthetic drug, a modified sulfa, related to vitamin B₁, known as thiazolyl (or phensulfazole or darvisul), developed by Murray Saunders, has been applied in Houston and Galveston, apparently with favorable results and no toxic effects. The drug is reported to reduce the fever in two days, as compared with the normal course of from five to eight days, and prevents the crippling effects.

Traditional treatment during the convalescent and chronic stages. The therapy technique subsequent to the subsidence of the acute symptoms used to include elaborate procedures such as: avoidance of fatigue or overstrain of the afflicted muscles; the use of radiant or nonradiant heat, to improve muscle

function; massage to improve circulation and nutrition, preferably given after the muscle has been warmed; support of the muscles in a condition of relaxation; avoidance of the effects of gravity and contractures (deformity) by various supports, such as splints, braces, casts, correctional jackets or corsets; a systematic program of muscular re-education to improve coordination, increase strength, and develop voluntary control, beginning with systematic, properly regulated passive and active movements of the palsied muscles, and proceeding to a comprehensive program of physiotherapy and occupational therapy adjusted to individual requirements. Hydrogymnastics, the movement of the limbs in warm water, is of value because the water counteracts the body weight and makes it easier for the child to execute the movements. The underwater treatment has been popularized by the hospital at Warm Springs, Georgia, established in 1927 by Franklin Delano Roosevelt who became a victim of the paralytic type of poliomyelitis in 1921 at the age of thirty-nine. This is the only establishment in the country devoted exclusively to the treatment of infantile paralysis. Incidentally, treatment for the physical rehabilitation of poliomyelitic and other kinds of crippled children is now available in public or private orthopedic divisions, hospitals, institutes, and Shriners' hospitals in all sections of the country. Heliotherapy or ultra-violet irradiation is of value for the general health improvement of crippled malnourished children.

A program of orthopedic surgery will be required at the appropriate stage by those suffering from severe degrees of paralysis that cannot be overcome by muscle training. The orthopedic treatment (in the narrow sense) varies with the case and may include tendon transplantation to restore muscular balance or prevent or correct deformities (when the muscular exercises have failed to restore sufficient muscle strength); tendon fixation; adhesive traction; fusion of joint surfaces; and bone lengthening or shortening.

The mechanical respirator (iron lung, designed by engi-

neer Philip Drinker in 1927) has prevented the deaths of thousands of polio victims suffering from severe paralysis of the muscles controlling respiration. Mechanical bellows force air into and withdraw it from a large airtight chamber in which the patient lies, with head projecting through a rubber collar, thus causing his chest to expand and contract. Those with chest paralysis receive the treatment at intervals for several weeks or months until the breathing muscles can function. Some persons with severe degrees of paralysis have remained continuously in the respirator for over 10 years. Fred Snite, Jr., of Miami Beach, has been dependent upon an iron lung since March 31, 1936.

More recently two or three types of portable cuirass respirators made of plastic or aluminum have been devised for temporary use until the tank (iron lung) type can be made available to the patient, but these inventions have not at this writing been approved by the Council on Physical Therapy of the American Medical Association. An electronic substitute has been devised to take the place of the mechanical respirator. An electrode attached through a small hole in the neck to the phrenic nerve restores respiration. The use of this device is still in the experimental stage.

The Kenny technique. In the unorthodox Kenny treatment,⁹ brought to the City Hospital in Minneapolis in June, 1940, by Nurse Elizabeth Kenny from the bush country in Australia, under the auspices of the National Foundation for Infantile Paralysis, the affected members are treated at the very beginning of the illness by hot packs which reduce the pain and muscle spasm and improve the circulation. Strips of heavy woolen cloth, cut to fit any part of the body, wrung out of boiling water to avoid blistering of the body and covered with waterproof material and a dry woolen cloth to preserve the heat, are placed over the painful member to relieve the hyperirritability, muscle spasm, and pain. The hot fo-

⁹ Kenny, Elizabeth, *The Treatment of Infantile Paralysis in the Acute Stage*. St. Paul: Bruce Publishing Company, 1941.

mentations are changed at intervals varying from a few minutes to about two hours. Although the treatment requires highly skilled nursing care, mothers can be trained to apply hot packs. The patient lies absolutely quiet on a mattress supported by a fracture board with the feet placed against a footboard in order to stimulate the proprioceptive sense from



FIG. 25. Treatment of infantile paralysis by means of the Kenny hot pack and passive and active movements.*

the standing reflex (see Fig. 25). The patient's position is changed frequently in order to keep him comfortable. As soon as the soreness and spasm have been removed and the joints can be moved passively without causing pain, the hands, fingers, feet, arms, and legs are moved gently by the nurse within a pain-free range. The patient is encouraged to attend to the moving limb, to visualize the movement, to think of executing it, and gradually to repeat the movement voluntarily. The muscle treatment is given twice daily. Little or

* Courtesy of George J. Boines, M.D.

no use is made of the immobilization technique. A recent improvement in the method consists in the administration of prostigmine by mouth or hypodermically several times a day, with or without the hot packs, to reduce the spasticity, incoordination, and deformities, increase the range of passive motion, and accelerate the recovery.¹⁰

Results of the combined Kenny and prostigmine treatments. The following results for a five-year period from the Kenny Clinic, established by George J. Boines, in the city communicable disease hospital in Wilmington, Delaware, the second Kenny Clinic in the United States, are suggestive.¹¹ In 1942 the Kenny packs replaced the orthodox method of treatment. In 1943 prostigmine was substituted for the packs. In 1944-45 packs and prostigmine were employed concurrently. The majority of patients treated, and also those most severely affected, came from the 1944 epidemic. Fifty-five of 88 in the 1944 group were paralyzed. Of 138 patients, who varied in age from ten months to forty-two years (83 per cent under fourteen), treated by the Kenny method and prostigmine (singly or simultaneously), 71.1 per cent were recorded as recovered, 10.1 per cent retained residual paralysis but were not incapacitated, 6.5 per cent could walk well with the use of an aid, 2.2 per cent were not able to walk without aid, and 5.1 per cent had died. The period of hospitalization had been reduced from an average of 160.2 days in 1941 (for the control group using the traditional methods) to an average of 32.1 days in 1945 for the Kenny-prostigmine group. The prostigmine could, in some cases, be substituted for the burdensome packs. As soon as the patients could walk they were released. "Approximately 75 per cent of patients with

¹⁰ Kabat, Herman, and Knapp, Miland E., "The Use of Prostigmine in the Treatment of Poliomyelitis," *Journal of the American Medical Association*, August 7, 1943, 989 f.

¹¹ Boines, G. J., "The Treatment of Poliomyelitis under Orthodox, Kenny, and Prostigmine Methods," *Delaware State Medical Journal*, 1946, 167-171; "The Use of Prostigmine and a Modified Kenny Technique in the Treatment of Poliomyelitis," *Journal of Pediatrics*, November, 1944, 414-438.

poliomyelitis can be adequately cared for at home without special nursing attention with this technique." Some chronic cases with deformities responded to the new treatment as well as some acute cases.

The Kenny-prostigmine procedure, apparently, greatly simplifies and shortens the treatment in all stages, and removes the necessity in many cases for permanent physical, educational, and social adjustments (see Fig. 26).

The educational program

The program of home or hospital instruction should begin for children of school age as soon as the child is declared ready for it by the attending physician. He should be entered in the regular or orthopedic school as soon as this is feasible with recommendations from the orthopedist concerning needed physiotherapeutic and orthopedic care. Children who require bus transportation and special services afforded by a properly equipped orthopedic school (or class) should remain in such a school as long as the special services are required.

All of the magnificent orthopedic day schools (such as those in Baltimore, Chicago, Cincinnati, Cleveland, Des Moines, Detroit, and many other places) have been erected during the present century and also all of the state hospital schools, with the exception of the Minnesota school which was established in 1897, and which provided an educational program from the start (although the program was not authorized by law until 1907). The typical day school is furnished with elaborate equipment of specially constructed chairs or adjustable chair desks (such as the American Universal desk), wheel chairs, treatment tables, tanks, pools, Burdick bakers, light therapy machines (ultra-violet, infra-red, diathermy), elaborate equipment for physiotherapy and for occupational therapy, and sometimes appliances for orthopedic treatment. The educational program, which usually parallels the work

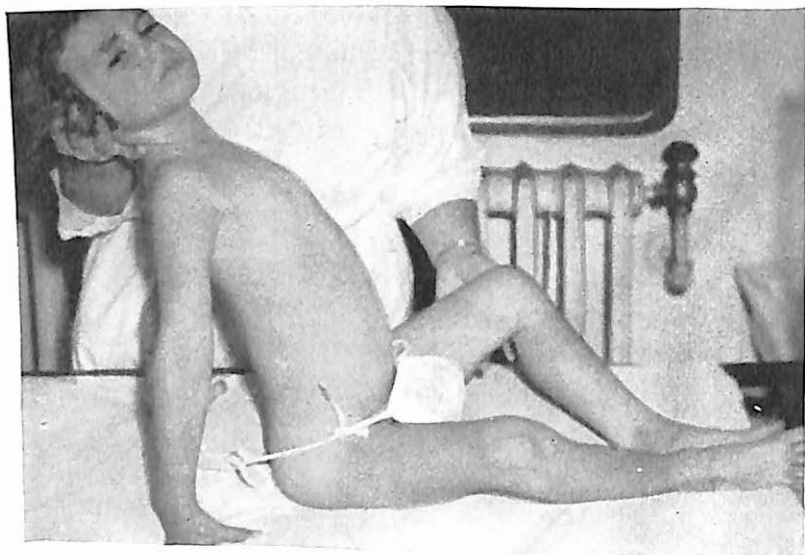


FIG. 26 (*Top*). P. P., age 6, at the time of admission to the hospital in the acute stage of poliomyelitis, five days after onset, unable to walk or stand on the right leg, marked rigidity in the nape of the neck and in the back, flaccid paralysis of the right leg and thigh, loss of adduction (movement toward median body line), flexion, and extension at hip, and contracted hamstring muscles. (*Bottom*) P. P., after being treated only one month and 18 days with prostigmine bromide 15 mg. tablets and prostigmine methyl sulphate ampules 1-2000 solution, without the use of the hot packs but with physiotherapy, chiefly muscle re-education according to the Kenny method. (Courtesy of George J. Boines, M.D.)

in the ordinary schools in the literary subject matter, often contains a rich offering of occupational activities and occasionally specific vocational training.

The distinctive features of the specially constructed school buildings for crippled children include, among other things, one-story construction, or two stories with properly constructed staircases, ramps, and elevators; the open court type of building; wide aisles with handrails along the walls and mirrors at the ends; rubber or cork floor coverings; absence of thresholds between the rooms; an examination room; rooms for muscle treatment and the adjustment of braces and casts; a curative gynasium; a curative workshop (physiotherapy); industrial art rooms (occupational therapy); a solarium for heliotherapy or for ultra-violet irradiation and rest rooms supplied with cots or reclining chairs.

Crippled children of subnormal mentality should be admitted without prejudice to classes for the mentally retarded if the school affords such classes, and should be retained as long as they require the physiotherapeutic treatments and can profit from them. Thereafter they should be transferred to classes for the mentally retarded in the regular school. In the event that the subnormals are denied admission to the orthopedic school, they should not be refused entrance on probation to a special class for the mentally deficient. Because of the limited number, such cases will not constitute a burden on any classroom. Crippled children of normal intelligence should be given every opportunity to advance in the standard academic curriculum, and when they no longer need the special physiotherapeutic services of the orthopedic school, they should be returned to the regular grades, unless, indeed, the orthopedic school affords needed opportunities for occupational or vocational training not available in the regular school. More opportunities should be afforded than has been the case in the past for vocational training suitable to the orthopedic condition and the vocational capability and proclivity of each child.

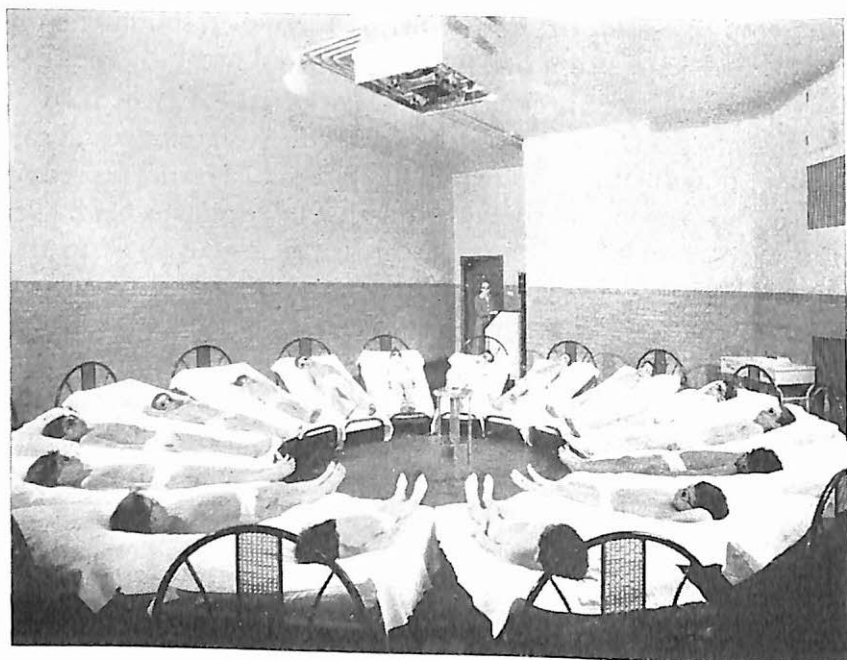
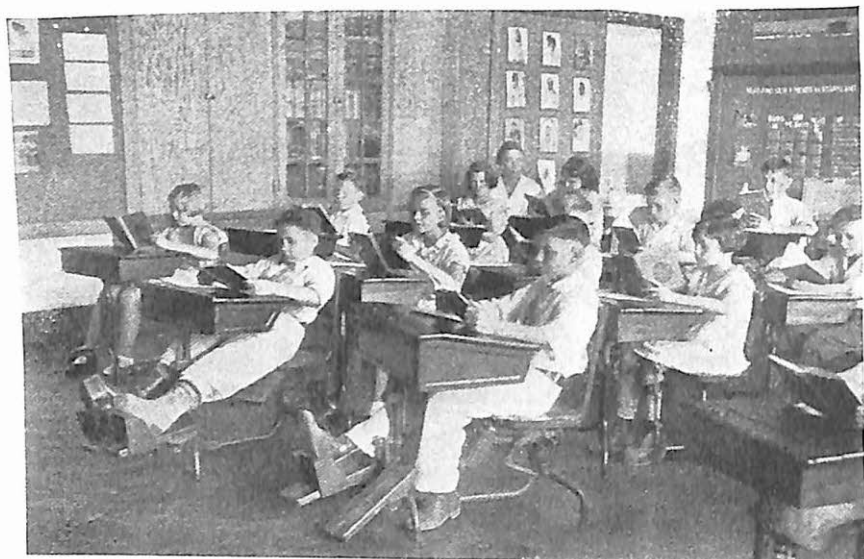


FIG. 27 (a & b) (Top). Crippled children seated at work in desk chairs especially designed for their comfort. (Bottom) Another group on an inclined platform is receiving ultra-violet radiation for general physical improvement. (Courtesy the William S. Baer School for Handicapped Children in Baltimore.)

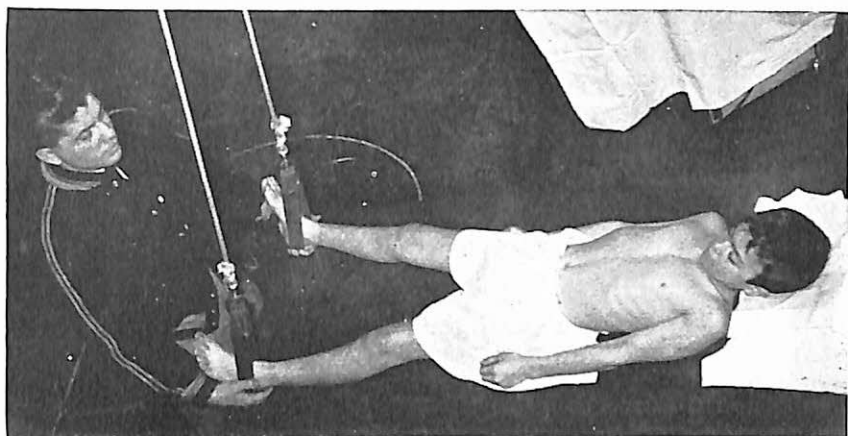


FIG. 27 (c). External rotation of the legs and hips to correct inward rotation of the feet due to muscular spasticity in a boy with cerebral palsy.*

Psychological treatment

Important throughout the entire course of the disorder are morale building and social rehabilitation. Judicious psychological treatment is needed to prevent or overcome the emotional problems created by long periods of hospitalization or immobilization, the feeling of lack of affection because of removal from home and friends, anxieties regarding growing deformities, and the like. Effective psychological treatment is also needed to avoid the crippling mental and social effects of pampering, indulgence, and overprotection on the one hand, and the discouragement and emotional conflicts engendered by discrimination, invidious comparisons, neglect, or cruelty, on the other hand. The aim should be to condition the child to assume a realistic attitude toward his problem, to face it frankly, unevasively, and without trepidation, to persevere without discouragement, and to develop self-reliance, initiative, and resourcefulness.

The crippled child often becomes a victim of self-pity and self-glorification, frequently because of the pampering and

* Courtesy of the Department of Special Education, Newark, New Jersey, Public Schools.

spoiling processes to which he has been subjected at home and in school. Unless wisely guided he may become a shirker or a tyrant and insist that the world owes him a living and that his every whim must be humored. The inability to compete on equal terms with other children in their physical plays and social activities may develop feelings of incompetency, seclusiveness, or dissatisfaction, or the determination to overcome the handicap and to excel in other directions.¹²

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¹² Wallin, *Personality Maladjustments and Mental Hygiene*, p. 174.

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Chapter 19

MICROCEPHALY

Deviations in head shape and size

Pronounced deviations from the typical head (mesocephaly) involve longheadedness, known as dolichocephaly (*dolikhos*, long; *kephale*, head) with a cephalic index¹ of under 77 for heads and 75 for skulls; broadheadedness, known as brachycephaly (*brakhus*, short), with a cephalic index of 80 or more; a high, pointed head, especially in the frontal region, with a vertical index above 77, and a short antero-posterior diameter, known as oxycephaly (*oxys*, sharp); acrocephaly (*akros*, point), steeple head, or tower head; a long, keeled or boatshaped head, flattened at the sides and converging upward to a keel, known as scaphocephaly (*skaphe*, boat); an abnormally tall and narrow head, known as leptcephaly (*leptos*, slender); and a wry, twisted, or asymmetrical head, known as plagiocephaly (*plagios*, oblique). These and other minor deviations in head shapes are of interest chiefly to the student of biological variation and anthropometry. The correlations of these head distortions with deviations in personality and intelligence are largely negative.² The student of mental and educational deviations should be particularly interested, however, in the characteristics of the microcephalic, hydrocephalic, mongolian, and macrocephalic heads

¹ Obtained by dividing the maximum breadth (transverse diameter) by the maximum length (antero-posterior diameter) and multiplying by 100. An index of 75 indicates that the width is 75 per cent of the length.

² For a discussion of these relations see Penrose, *Mental Defect*, pp. 22-33, and 128, and the references listed.

as part of the symptom-complex of microcephaly, hydrocephaly, mongolism, and hypertrophic sclerosis. The macrocephalic, cretinous, mongolian, rickety, achondroplastic, and hydrocephalic heads are described on pages 249, 273, 288, 311, and 502 f.

The microcephalic head

The diagnosis of microcephaly is based on two essential characteristics affecting the size and shape of the head. The maximum circumference at maturity ordinarily does not exceed 17 to 18 inches. The head girth is considerably less than that of the chest, although the opposite is the case in normal infants. The shape of the head is also characteristic. Typi-



FIG. 28. J. C., microcephalic idiot, age 29.4, Stanford-Binet age 1.5, head circumference at age 10, 16.5 inches.*



FIG. 29. W. P., microcephalic idiot, age 54.7, Kuhlmann-Binet age 1.9, head circumference at age 42, 16.5 inches.**

* Wallin, J. E. W., *The Education of Handicapped Children*. Boston: Houghton Mifflin Company, 1939.

** Courtesy of E. Arthur Whitney.

cally the head is long and narrow, with a low cephalic index of about 75, a rapidly retreating forehead, a flattened occiput, and a vertex that tends to be pointed (see Figs. 28 and 29). The face often appears large in contrast to the diminutive head, although it may not be larger than that of a normal person. Ordinarily the anterior fontanelle and sutures are closed at birth or close very soon thereafter. The redundant skin on the head is sometimes furrowed in the direction of the antero-posterior diameter. The scalp is often covered with very stiff and coarse hair.

Some heads classified as microcephalic with respect to shape are borderline or transitional so far as size is concerned. Tredgold records several "typical microcephalics" with head circumferences of 19 inches or more and one with a head girth of 21 inches. The head girths of Benda's 19 microcephalics, ages one to thirty-two, vary from 12 to 21 inches, all except two being under 19 inches. The girths of the heads in Figs. 28 to 30 are 16.5 and 18 inches. The brain weights have varied from 4.7 ounces (a cystic degeneration case of Benda's) or 6 ounces (Sander's case) to about 31 ounces (one of Benda's developmental cases without accumulation of spinal fluid).³ The brain of the publicized Freddy, shown in Plates III and IV, opposite pages 59 and 60, in Shuttleworth and Pott's *Mentally Deficient Children*, weighed 12.5 ounces. His maximum cranial circumference at the time of death at twenty-nine was 15 inches.

The significance of these measurements becomes apparent when they are compared with the corresponding measurements of normal infants at birth or a few days thereafter,⁴ which are, for males about 12 ounces and 14 inches. At twelve months the corresponding measurements are 32 ounces and 18 inches. In other words, the maximum cranial measurements of the adult microcephalic are ordinarily about on a par with those of a one-year-old child.

³ Benda, "Ten Years Research in Mental Deficiency," p. 176.

⁴ Tredgold, *Mental Deficiency*, pp. 530-532.

The nature of the brain pathology

Added to the diminution of the size and weight of the microcephalic brain is a host of pathological brain tissue changes. These include diminution in the number and non-development (agenesis) or imperfect development of the neurones (neuroblasts), particularly in the parietal and occipital lobes of the cerebrum; smallness of the convolutions (microgyria); adhesion of the brain membrane; areas of atrophic sclerosis; cystic degeneration; hemiatrophy; porencephaly; and external or internal hydrocephaly. Not all of these neurological defects exist in all microcephalic brains. About 15 per cent of post mortems reveal evidences of porencephaly and hydrocephaly. The cerebellum usually remains intact. In the cases of developmental failure the developmental arrest, according to some authorities, occurs at about the fourth or fifth fetal month. In the degenerative cases the white matter is destroyed before the gray cells. The spinal cord is frequently involved also, usually to a greater extent than the cerebellum.

Characteristics

The body of the microcephalic. Although the body may be well developed and free of marked abnormalities, many microcephalics are dwarfish in stature, attaining a height of only five feet or less. Some microcephalics, especially among the lower grades, are subject to cerebral spasticity or various degrees of paresis, particularly in the lower limbs. Stiffness of limb is a common characteristic. The gait may be noticeably unsteady, even in the absence of paralysis. About 50 per cent are subject to epileptic seizures. Although relatively free of sensory impairments, some suffer from optic atrophy and nystagmus.

Psychological characteristics. Several attempts have been made to relate the degree of mental defect to the size of the brain as indicated by the head circumference. For example:

	<i>Idiocy</i>	<i>Imbecility</i>	<i>Feeble-mindedness</i>
Tredgold ^a	No more than 13 inches.	No more than 17 inches.	2 inches below normal. ^b
Shuttleworth ^c	Under 18 inches.	18 to 19 inches.	19 to 20 inches. ^d
Voisin ^e	11 to 13 inches.	14 to 17 inches (too small for ordinary intelligence).	18 to 18.5 inches; "may permit of intellectual processes."

^a Tredgold, *Mental Deficiency*, p. 115.

^b "Probably . . . feeble-mindedness at least."

^c Shuttleworth and Potts, *Mentally Deficient Children*, p. 138.

^d "Not an uncommon measurement in cases of mere feeble-mindedness."

^e Quoted from Barr, M. W., *Mental Defectives*, pp. 204, 208. See Barr's comments (p. 206) on artificially produced microcephaly by head binding by Slavs, Anglo-Saxons, Celts, Peruvians, some North American Indian tribes, and others.

Although numerous more exact investigations⁵ have revealed a positive correlation between head size or cranial capacity and intelligence as determined by tests, the relationship is not sufficiently exact to be of much value for purposes of diagnosis. A small head is not incompatible with high intelligence nor is a large head incompatible with feeble-mindedness. The mean brain weight of the ancient Peruvians, who attained a high cultural development, was only 40.1 ounces (Tredgold). Edward Seguin relates that he saw Aztec children (the Aztecs founded Mexico) "whose heads were under 13 inches in circumference" who "could have been educated like human beings."⁶ Shuttleworth and Potts "have repeatedly seen boys and girls with heads measuring only 19 inches taught to read and write and do industrial work."⁷ Some microcephalics with head girths of from 15 to 17 inches have evidenced more practical ability than could have been surmised from the head size.⁸ Among my early St. Louis cases was a child with a diminutive head (about 19½ inches) whose father, a successful cigar salesman, also had a very small head, neither with the microcephalic shape characteristics,

⁵ For a summary and references consult Penrose, *Mental Defect*, pp. 25-32.

⁶ The "Aztec" boy and girl (of American-Indian origin), exhibited in Europe and America for years and publicly married in London in 1867, could repeat only a few isolated words, were very vivacious, excitable, and curious (Tredgold, 227), and were diagnosed as idiots (Barr, 207).

⁷ Shuttleworth and Potts, *Mentally Deficient Children*, p. 60.

⁸ For illustrative cases, see Tredgold, *Mental Deficiency*, pp. 226-228.

however. Some great intellects have had small heads: René Descartes, Percy Shelley, Fascola, Schumann, Gaetano Donizetti, Anatole France (39 ounces), and Leon Gambetta (40.9 ounces).

Although more small heads are found among feeble-minded than among normal children—Fraser found 27.5 per cent among “morons” as against 8.3 per cent among normal children—the brains of the feeble-minded occasionally exceed the brain weights of superior normals. Thus the maximum brain weight was 55 ounces among 100 male “imbeciles” and “idiots” (Edward B. Sherlock). The brain weight of one feeble-minded boy of fifteen was 62 ounces, 9 ounces heavier than Napoleon’s brain and only one ounce less than Daniel Webster’s. Most of the heavy brains among the mentally defective are probably cases of hypertrophic sclerosis.

Brain weight should, however, be considered in relation to body weight. Thus, though the horse’s brain is over three and a half times heavier than man’s brain, in proportion to body weight man’s brain is about ten times heavier. The proportion of man’s brain weight to body weight is 1 to 46 (Dubois), but curiously the ratio for 15 idiots “selected at random” has been given as 1 to 34.

The inescapable implication of the data presented is that intelligence is not very closely related to mere brain size. It is doubtless much more closely related to the quality than to the quantity of the nervous tissue of the cerebrum, more particularly to the fineness of organization and the sensitiveness or responsiveness of the neurones in the cortex.

Nearly all microcephalics contacted by the writer in institutions, special classes, and in his psychoeducational clinics have been low grade mental defectives. Barr classified most of his cases as idiots. Shrubsall and Williams, though finding “any grade of mental deficiency” among microcephalics, classify the majority as imbeciles.⁹ Tredgold classifies a “considerable number” as idiots, helpless, unable to speak, and

⁹ Shrubsall and Williams, *Mental Deficiency Practice*, p. 52.

able to understand only a few words. "The majority" are imbeciles, able to speak only a few words or formulate only simple phrases, and perform only simple tasks, but able to understand most that is said to them. "A few" are merely feeble-minded (morons) who can "earn their living under supervision."¹⁰ Most of the higher grades are probably borderline cases from the standpoint of head size.

Although it is true that the intelligence of microcephalics is sometimes underestimated because of the smallness of the head, it is equally true that it is often overrated because of the quickness of their reactions, which lends a picture of superficial brightness. Although the microcephalic is observant and imitative, he is difficult to teach, not only because of his low mentality but also because of his limited attention span and high distractibility.

The typical microcephalic improves slowly until he reaches a premature arrest. Only a few deteriorate mentally during the growth period. Those who live long may deteriorate mentally as a result of senile changes.

The microcephalic tends to be restless, excitable, vivacious, and quick in his reactions like a bird or a monkey. The quick and restless movements and the narrow face, cone-shaped head, and receding chin led Cesare Lombroso in 1873 to refer to some microcephalics as "bird men," "rabbit men," and "geese men." A certain number of them constitute genuine cases of psychic infantilism. Usually amiable and well-behaved, the microcephalic may be subject to unprovoked, passionate outbreaks and may at times become troublesome and quarrelsome.

Education of microcephalics

All except the lowest grades or the most unstable are amenable to stabilization and conduct improvement. In the training of microcephalics the emphasis should be placed on habit formation for the purpose of self-care and practical use-

¹⁰ Tredgold, *Mental Deficiency*, p. 225.

fulness, on the development of manual skill, and on socialization or habit training directed toward obtaining social conformity and curbing antisocial or unsocial proclivities. The academic rudiments should not be denied the few who can profit from literary instruction. The exclusion of stable, high grade microcephalics from public school special classes merely because of the size and shape of the skull should not be countenanced. The number of such cases will be relatively small. Penrose places the number of microcephalics at "scarcely 1 per cent" of institutional cases, and Tredgold at not over 0.5 per cent of aments of all ages (many microcephalics die early) and not over 6 per cent of those under age ten. If transitional or borderline head sizes and shapes are included, the number would be increased appreciably.

Causation

Craniectomy, excision of a linear strip (see Fig. 30), to allow the brain to expand, beginning in 1878 in Montreal, Canada, and practiced about 1890 in the United States (especially at the Elwyn Training School in Pennsylvania),¹¹ France, and England, based on the assumption that the brain had failed to grow because of the premature ossification of the anterior fontanelle and sutures, has been abandoned because of its demonstrated futility. The small skull is not the cause of the small brain, but is merely molded to the brain. Head operations may be justified when evidence exists of pressure or brain irritation from operable localized lesions or cysts. But the operations must be performed before degenerative changes occur.

The atavism theory of C. Vogt, of Geneva, based on superficial physical and behavior resemblances, that microcephaly is a reversion to a prehuman or simian type has also been abandoned. Many apes are superior to many microcephalics in ability and shrewdness.

Microcephaly has usually been considered as an expression

¹¹ See Barr, *Mental Defectives*, pp. 182-188.

of germinal blight or morbid inheritance. Cases of familial incidence of the disorder lend credence to this theory. Among such cases are Houze's two microcephalic siblings, with a microcephalic father and grandfather; and Erik J. Larsen's five cases of microcephaly with several microcephalics in the family.¹² Lapage reports six microcephalics among eight siblings, and Bernstein reports four male and one female

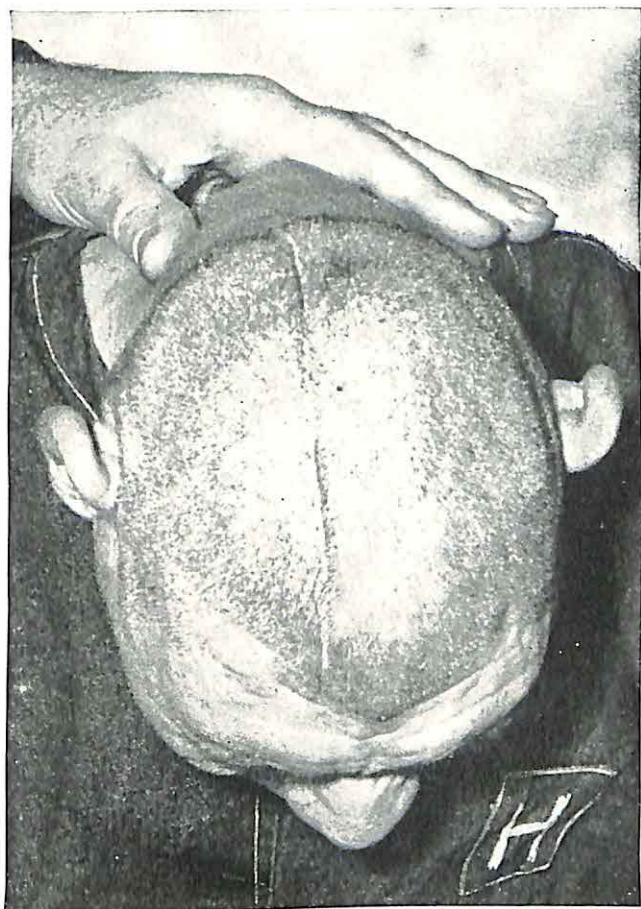


FIG. 30. H. H., helpless microcephalic idiot, whose head shows a groove seven and a half inches long from a craniectomy operation in infancy. Head circumference at age 44 almost 18 inches.*

¹² Tredgold, *Mental Deficiency*, pp. 222 f.

* Courtesy of E. Arthur Whitney.

microcephalics among ten siblings of mentally normal parents.¹³ In commenting on a recent pedigree chart of two male and one female microcephalics in three generations, Allan, Herndon, and Dudley cautiously remark that "this family represents the action of a gene which in simplex state merely renders the fetus particularly susceptible to adverse influences during intrauterine life."¹⁴ On the other hand, Halperin is inclined to accept Penrose's assumption that microcephaly in familial cases without any discoverable pathogenic factors is caused by a recessive gene. He cites three such pedigrees: three microcephalic children in a sibship of five; three girls, the last born of eight sibs—two miscarriages—(see Fig. 31); and a boy and a girl in a sibship of thirteen (one stillbirth).¹⁵

Greenfield and Wolfsohn have attributed the condition to the action of metabolic toxins or parental alcoholism on the genes before conception or to some pathological process that affects the fetus, such as infection from meningitis or encephalitis.¹⁶ Benda ascribed 50 per cent of his cases to "prenatal developmental anomalies and cystic degeneration of the brain." The degenerative cases are characterized by cavities filled with fluid. Some of the latter may have been caused by fetal encephalitis. The other cases, apparently, were due to failure of "brain development."¹⁷

It is now known that many cases of microcephaly are of exogenous origin. The clearest evidence of externally produced microcephaly comes from therapeutic pelvic irradiation with X-rays or radium of pregnant mothers who have

¹³ Bernstein, Charles, "Microcephalic People Sometimes Called 'Pin Heads,'" *Journal of Heredity*, January, 1922, 30-39.

¹⁴ Allan, William, Herndon, C. Nash, and Dudley, Florence C., "Some Examples of the Inheritance of Mental Deficiency: Apparently Sex-Linked Idiocy and Microcephaly," *American Journal of Mental Deficiency*, April, 1944, 325-334. (Contains comments on various rare types of mental deficiency, with bibliography.)

¹⁵ Halperin, S. L., "Three Pedigrees of Microcephaly," *Journal of Heredity*, July, 1944, 211-211.

¹⁶ Greenfield, J. Godwin, and Wolfsohn, Julian M., "Microcephalia Vera," *Archives of Neurology and Psychiatry*, June, 1935, 33:1296-1316.

¹⁷ Benda, "Ten Years Research in Mental Deficiency," pp. 174 f.

suffered from some disease. Murphy reports that 14 of 53 fetuses from mothers irradiated with X-rays during pregnancy were microcephalic and 13 were otherwise abnormal, including two mongols, one case of spina bifida (cleft spine), and one with a deformed skull. Nine were aborted. On the



FIG. 31. Three microcephalic siblings, the last of a sibship of eight. There seems to be a hereditary basis for the deviation; no predisposing causes on the part of the mother could be found.*

other hand, of 256 babies born of mothers subjected to pre-pregnancy irradiation, only one was microcephalic, nine were otherwise abnormal, and 67 abortions occurred.¹⁸

Goldstein and Murphy report that 106 irradiated women delivered 74 full-term children. Thirty-eight of the children showed some kind of defect, the most common of which (16 cases) was microcephalic mental defectiveness.¹⁹ Goldstein

¹⁸ Murphy, Douglas P., "Ovarian Irradiation: Its Effect on the Health of Subsequent Children," *Surgery, Gynecology, and Obstetrics*, 1928, 47:201-215.

¹⁹ Goldstein, Leopold, and Murphy, D. P., "Microcephalic Idiocy Following Radium Therapy for Uterine Cancer During Pregnancy," *American Journal of Obstetrics and Gynecology*, 1929, 18:189-195.

* Halperin, Sidney, "Three Pedigrees of Microcephaly," *Journal of Heredity*, July, 1944, 35, 7: 211.

in a later study reveals that 20 of 76 children irradiated in utero manifested marked defects, 19 being microcephalic.²⁰ Radiation of the brains of young animals has resulted in similar birth defects. X-ray treatment, obviously, interferes with embryological development, particularly of the brain. The cessation of this kind of treatment will prevent the birth of many microcephalics. An effective eugenic program for the prevention of familial microcephaly cannot be formulated as yet because of our imperfect knowledge of the genetic factors.

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Maxfield, Francis, "A Case of Microcephaly Following Prenatal Roentgen Irradiation," *American Journal of Mental Deficiency*, January, 1941, 358-365.

ILLUSTRATIVE CASES: J. C. (Fig. 28) is a microcephalic idiot, age 29.4 when photographed, Binet age 1.5 at the age of twenty-nine. Head circumference 16.5 inches at age ten. Lower jaw prognathic, large mouth, hair on upper lip, eyes small and slanting. One brother a microcephalic idiot, and one cousin feeble-minded. Walked at three. Masturbates. Tends to mutilate hands and other parts of body, often rough with other children, playful, laughs without provocation, makes quick birdlike movements of the head, likes to show off. Capable of matching two colors, of selecting common objects by sense of touch, of dressing and undressing herself, although requiring assistance in lacing shoes, of carrying clothing from one place to another and of carrying out one simple command, such as "get up" or "sit down."

W. P. (Fig. 29), "a perfect type of idiot," was admitted to the Elwyn Training School at the age of 6-10, and was 5-4-7 when the photograph was taken. At the age of 42-6 his head circumference was 16½ inches, cephalic index 77, height 5 feet, weight 105 pounds, and Kuhlmann-Binet 1-9, I. Q. 10.9. Often incontinent, and frequently has to be dressed. Speaks a few words, but his speech is almost unintelligible. Used to laugh or weep without

²⁰Goldstein, L., "Radiogenic Microcephaly," *Archives of Neurology and Psychiatry*, 1931, 24:102-115.

cause. His movements are jerky. Cooperative and helps a little in dry scrubbing, but "likes to be sick" and always has a pain. Has a violent temper, is quarrelsome, an agitator and frequent fighter. Likes to show off. Incapable of concentration.

H. H. (Fig. 30) was admitted at the age of six to Elwyn with his twin microcephalic brother (who died in the influenza epidemic in 1918). Age fifty-six when head was photographed. Head shows a longitudinal groove from a craniectomy operation in infancy, which proved barren of any good. At the age of forty-four, head girth almost 18 inches, cephalic index 82, height 63 inches, and weight 106 pounds. Difficult birth; mother ill for four years. A "helpless idiot" who has to be dressed but who speaks a few words and understands many things said to him. Used to "amuse others by turning somersaults quickly." "Looks like a monkey." For years has bitten himself and torn off clothes at times. Sways and holds head forward.

Chapter 20

HYDROCEPHALUS (OR HYDROCEPHALY)

Normally the cerebrospinal fluid, possibly a biological filtrate, flows from the choroid plexuses (folds of the pia membrane in the two lateral and third and fourth ventricles) where much the greater part is produced, through the cerebrospinal circulatory system to the subarachnoid space (next to the pia, the innermost membrane of the brain and the spinal cord), where it is absorbed. The fluid flows through a series of canals from the lateral ventricles to the third and fourth ventricles and then to the spinal subarachnoid space (about one-fifth of the fluid) and the cerebral subarachnoid space (mainly through the Sylvian aqueduct). About 60 per cent of the fluid is absorbed directly into the small blood vessels or the arachnoid villi (small absorbing processes) in the cerebral subarachnoid space. Normally the fluid is replaced in the ventricles every six to eight hours, according to Michelson,¹ and in the cerebral subarachnoid space five or six times every 24 hours, according to L. B. Flexner.²

Basic nature and genesis of the disorder

This chronic condition, popularly referred to as "water on the brain," is caused by the accumulation of the cerebrospinal fluid in the spaces outside the cortex between the dural (outermost) and the arachnoid (middle) membranes (the

¹ Michelson, Jost J., "Surgical Aspects of Cranial and Intracranial Malformations," *American Journal of Mental Deficiency*, July, 1913, 15-32 (52 references).

² Bucy, Paul C., "Hydrocephalus," in *Practice of Pediatrics*, Chap. 3, pp. 1-28 (62 references).

external form) or in the internal cavities known as the ventricles (the ventricular or internal form).

The internal variety is classified as "obstructive" when the fluid is prevented from leaving the ventricles through an obstruction in the pathways and as "communicating" when the ventricular fluid can reach the spinal subarachnoid space but cannot reach the cerebral subarachnoid space (Walter E. Dandy).³ The latter is the most prevalent type of hydrocephalus. The internal form is far more prevalent and menacing than the external form, which is secondary to the internal. In the internal form the fluid accumulates in the cavities when the interconnecting channels are blocked by congenital malformations, by tumors (neoplasms), or by lesions (glia or scar tissue) produced by inflammatory processes from infectious diseases, such as generalized or localized (basal) meningitis, meningitis of sinus or otitic (ear) origin,⁴ by congenital syphilis, or by intracranial hemorrhages from birth injuries. The degenerative inflammatory process affects among other parts, the choroid plexus and the lining membrane (ependyma) of the ventricles. Protrusion of the meninges affects a certain proportion of cases. Hydrocephalus may begin during almost any period of life, but the large majority of cases originate in prenatal or early postnatal existence. In the infantile form the condition frequently is apparent at the time of birth or shortly thereafter. In the case of congenital syphilitics it ordinarily becomes apparent between the fourth and ninth months. About one-third of congenital syphilitic infants develop hydrocephalus (P. C. Jeans and J. V. Cooke).

³ For a more detailed description and a diagram of the paths of conduction for the cerebrospinal circulatory system see Louttit, *Clinical Psychology* (revised edition), pp. 205 f.; and for a more detailed explanation of the obstructive and communicating types see Sherman, *Intelligence and Its Deviations*, pp. 146 f.

⁴ The term otitic hydrocephaly was suggested by C. P. Symonds in 1931 and the term otorhinogenic by Ernest Reeves in 1941. See Votow, Robert E., "Otorhinogenic Hydrocephalus," *The Annals of Otology, Rhinology, and Laryngology*, December, 1944, 679-687 (14 references).

According to Dandy, occlusions in the aqueduct of Sylvius (a canal or tube from the third to the fourth ventricle), leading to the accumulation of fluid in the two lateral and third ventricles, are responsible for 50 per cent of the hydrocephalus in infancy, about 95 per cent of them being congenital. After infancy the obstructive internal form is rare, tumors being responsible for "perhaps 95 per cent" of the cases. "In children the most common lesion causing intracranial pressure is a tumor, and about two-thirds of these tumors are in the cerebellum."⁵

The blocking of the ducts or the failure of absorption of the fluid results in endocranial pressure and numerous physical and mental symptoms, depending on the age of onset, the location of the lesion, and the degree of interference with the circulation of the fluid. The occlusion may vary from total to partial interruption of the circulation. However, in the case of the external form great intracranial pressure does not ordinarily result.

Physical signs and symptoms

One of the most conspicuous early signs of severe internal hydrocephaly that develops in early life, before the ossification of the cranial suture (line of junction between adjacent bones) has taken place, is the gradual enlargement of the head in all directions from the constant pressure of the dammed-up fluid against the adjacent brain tissue. Distension of the ventricles, above the point of the obstruction, causes a flattening of the brain against the skull cap and a compression of the optic nerve. The intracranial pressure, which may cause the cerebral tissue to become very thin or disappear entirely, is against the vault of the skull rather than against the base. The head, top-heavy in appearance, tends to become globular, balloon-like, or pyramidal in shape, with the greatest circum-

⁵ Dandy, Walter E., "Diagnosis and Treatment of Strictures of the Aqueduct of Sylvius (Causing Hydrocephalus)," *Archives of Surgery*, July-August, 1945, 1-14.

ference at the level of the temples.⁶ The expansion is particularly apparent in a bulging forehead, a bulging, tense, and enlarged fontanelle, separated sutures, and protrusion at the root of the nose. The bones may become thin. The sutures eventually close by the development of wormian bones. As the skull expands, the skin becomes tightly stretched, so that the blood vessels may stand out prominently, and the skin may rupture, allowing the fluid to escape. The eyes are often pressed downward or the lids elevated, exposing the upper sclera (white coat) of the eyeball, or the eyes may appear to protrude. In contrast with the large head, the face appears small and V-shaped. Sometimes percussion of the head produces a "cracked-pot sound," which cannot be elicited after the head fills up with fluid. The head enlargement may proceed very rapidly in severe early-life cases or very slowly and gradually for several years in cases of partial occlusion.

Tredgold gives the average head girth of institutional cases as about "25 or 26 inches," with a maximum of "30 inches or more."⁷ The measurements of 29 of Dandy's cases from one year to forty-five years of age (only four over twenty) varied from 23.2 to 25.6 inches. The size of the ventricles for 11 cases, based on the amount of fluid needed to fill them, varied from 8.6 to 44 cubic inches.⁸ Ten of Benda's cases varied from 17.5 to 35.12 inches.

The following are the measurements of the three largest hydrocephalic heads presented in clinics conducted for my Duke University students at the Casswell Training School in North Carolina:⁹

⁶For a description of the shape of the hypertrophic head see p. 249. In rickets the head, often enlarged from thickening, is commonly asymmetrical, with prominent bosses and ridges, but without prominent veins. The fontanelle if open is depressed instead of distended.

⁷Tredgold, *Mental Deficiency*, p. 267.

⁸Dandy, *op. cit.*, pp. 9 f.

⁹The psychological data were supplied by Elizabeth M. Brown, psychologist at the institutions.

For B. Myers's and Bird T. Baldwin's measurements of the head sizes of normal infants and children see Bucy, "Hydrocephalus," in *Practice of Pediatrics*, p. 17.

At Time of Clinic:

<i>Case</i>	<i>Sex</i>	<i>Binet I.Q. at</i>	<i>Chron. age at time of clinic</i>	<i>Supra- orbital Head Girth</i>	<i>Maximum Head Girth</i>	<i>Comments</i>
B.P.	M	41 at 6	11	27.5 in.	30 (over upper fore- head)	Blind. "May not be true measure of intelligence." Died at 12-7.
B.L.	M	32? at 17-10	16	29.5 in.		Bedridden, deformed body; arms, legs, hands rigid; unable to hold up big head; imbecile; died at 19-6.
J.F.J.	M	26? at 7	8	26 $\frac{3}{8}$ in.	27 $\frac{5}{8}$ (over upper fore- head)	Talked well and clearly; 6 months before death less animated, took less inter- est; eyes affected. Died at 11-11.

It is obvious that appreciable head dilation cannot occur even in severe cases when the disease begins after the cranial bones have become fully hardened and the sutures have become ossified. Hydrocephalus may be present nevertheless. Post mortem examinations have revealed that hydrocephaly is a rather common complication even in microcephalic brains, due, perhaps, to hypoplasia of the hemispheres.

The following are among the traditional signs of hydrocephalus which aid in its diagnosis in heads of normal shape and size. These include periodic attacks of headache and vomiting (one of the most persistent early signs); the development of visual defects, such as photophobia, strabismus (squint), nystagmus, optic neuritis or choked disk (or "papilledema," present in 40 per cent, according to Votow), and blindness; deafness; development of a stiff neck, retracted head, and paralysis (usually of the legs, such as hemiplegia or paraplegia¹⁰); development of epileptic convulsions (espe-

¹⁰ Yakovlev, Paul L., "Paraplegias of Hydrocephalics," *American Journal of Mental Deficiency*, April, 1917, 561-576.

cially in the acute stage and in the terminal stage of the progressive type); restlessness, irritability, and fussiness; slowness in learning to walk or talk; feeding difficulties and malnutrition. Many of these symptoms are caused by the great intracranial tension. In the most extreme cases the destruction of the brain tissue is so complete that only a bag of fluid remains surrounded by a thin layer of cortical cells in which the convolutions may be atrophied. Convolutional atrophy is almost pathognomonic of hydrocephaly. Early diagnosis of the degree and type of the malady is now possible in many cases, however, by means of the X-ray or the pneumoencephalogram,¹¹ which reveal the location and size of the edematous cavities, the presence of convolutional atrophy, and the height of the occipital protuberance (inion) which is pushed upward by cerebellar tumors. The "dye test" (based on the indicator, phenol red, injected into one lateral ventricle) is used to reveal the presence of an obstruction in the ventricles. If the dye passes from the ventricles into the spinal cord, the blockage is not in the ventricles but in the reservoirs (cisternae) at the base of the brain (giving rise to communicating "hydrocephalus").¹²

Prevalence of hydrocephaly

More recent investigations by the newer methods seem to show that more severe cases, and especially mild cases without pathognomonic signs, exist than was suspected earlier. In England years ago Lapage found no hydrocephalics among 100 consecutive hospital defectives of all grades, and Still reported only 0.28 per cent among 350 defectives. Among 904 candidates for special class assignments in Manchester, England, as reported by Lapage, 3 per cent of the rejects (low grades) and 0.25 per cent of those admitted were hydrocephalics. The corresponding percentage among 900 consecutive examinees in the St. Louis psychoeducational clinic, all

¹¹ An X-ray of the ventricles (ventriculogram) completely filled with air.

¹² Dandy, "Strictures of the Aqueduct of Sylvius."

examined by the writer, was 0.4 per cent (four cases, two feeble-minded), constituting 0.9 per cent of the imbeciles and 0.6 per cent of all the morons.¹³ The screening of many of these hospital and clinic cases by the X-ray procedures now in vogue would, doubtless, have disclosed many more hydrocephalics. This is suggested by the studies of Felix Frisch on mild or obscure forms of hydrocephalus which he designates "hydrocephalus occultus."¹⁴

Mental sequelae

Hydrocephalics differ markedly in mental capacity and in personality characteristics. Many are normal or bright and remain so if the pathological condition becomes stationary or clears up through spontaneous recovery or curative treatment before permanent damage has been done to the nerve cells. In some, however, the hydrocephalus is superimposed upon a background of permanent primary mental deficiency. Some of the congenital cases are probably the product of defective intrauterine conditions or accidents of prenatal development rather than of defective inheritance. At any rate, the familial incidence of the anomaly is very infrequent. Most cases of mental defect and retardation among hydrocephalics (largely confined to the internal form) are secondary to the neuronc degeneration produced by the fluid tension. All degrees of mental subnormality may exist from profound idiocy to a "slight degree of mental impairment." The mental status among both normal and subnormal cases, as well as the physical condition, is likely to deteriorate in the severe unchecked progressive type of affliction until the patient becomes bedridden and succumbs, ordinarily within three or four years, from the devastating effects of the malady itself or from some intercurrent disease. No cure exists for the mental defectiveness thus produced if the nerve cells have

¹³ Wallin, "The Diagnostic Findings."

¹⁴ Frisch, Felix, "Psychological Observations in Cases of Hydrocephalus Occultus," *American Journal of Mental Deficiency*, July, 1941, 52-54.

been permanently blighted or destroyed. Although the degree of mental defect is roughly proportional to the amount of cerebral destruction, many exceptions occur. The degree of mentality that some hydrocephalics have evidenced in life has been quite incompatible with the neural ravages disclosed by post mortem examinations or by electroencephalograms.

Felix Frisch believes that mild or obscure forms of hydrocephaly account for various "psychopathic states" or behavior anomalies in children, such as spells of ill-humor, sullenness, and rapid, unprovoked changes of disposition ("an almost infallible sign of increased endocranial pressure"). Such children fatigue very rapidly regardless of the nature of the activity in which they are engaged, become inattentive, and begin to yawn. The pneumoencephalograms of these children, who often possess unusual memories for numbers, reveal a moderate general dilation of the ventricles.¹⁵

Most hydrocephalic children coming under the writer's observation, mostly high grade mental defectives, have been quiet, well-mannered, friendly, cooperative, and fairly dependable, but lacking in physical and mental vigor and in persistence, and have been slow, awkward, or clumsy in movement.

Preventive and curative medical treatment

Various treatment procedures have been tried with varying degrees of success. The administration of thyroid extract, potassium iodide, or diuretin, as sorbefacients for absorbing the excess fluid, or dehydration by limiting the fluid intake or otherwise, are of questionable efficacy. Recently Simon Stone has reported favorable results from the administration of vitamin E (wheat germ oil) in the case of nine children with the congenital nonobstructive type without complications. "In all the treated patients arrest or marked slowing

¹⁵ Frisch, "Cases of Hydrocephalus Occultus." For a recent psychological study of hydrocephalics see Teska, P. T., "The Mentality of Hydrocephalics and a Description of an Interesting Case," *Journal of Psychology*, April, 1947, 197-203.

down of progression of hydrocephalus occurred."¹⁶ In fact, hydrocephalus did not develop even in two of five patients in whom meningoceles (hernial protrusion of the meninges) were removed during medication. Good results from anti-syphilitic treatment of congenital syphilitics have been reported by Jones and Cooke.

Many operative procedures have been tried for many years. One of the most radical procedures, allegedly successful, was reported in the press in 1942. It consists in the removal from the infant skull of strips of bone four-fifths of an inch wide on either side of the sutures (seams) which allows the brain to expand. The excised slits eventually become filled with bone. Eighteen years after the reported operation on the infant, the skull was of normal shape and the "individual was rated as having perfect vision and superior intelligence." More evidence is needed in support of the value of this dubious procedure. Favorable results from the coagulation of the choroid plexus for the purpose of reducing the secretion of the cerebrospinal fluid have been reported by Putnam, but fatal results sometimes follow the operation.¹⁷

Withdrawal of the cerebrospinal fluid by means of the lumbar puncture technique gives only temporary relief—say, from 24 to 48 hours—and the operation must be repeated at intervals. Detouring the fluid by means of a buried rubber tube from the lateral ventricle to the subarachnoid space under the cerebellum (the method of Torkildsen, 1931) introduces complications from the presence of a foreign body in the organism.

Apparently the best operative method for removing the stricture of the aqueduct is that devised by Dandy. This consists in making an aperture in the floor of the third ventricle "from which the fluid can be sidetracked from the di-

¹⁶ Stone, Simon, "Wheat Germ Oil (Vitamin E) in the Treatment of Congenital Non-Obstructive Hydrocephalus," *Journal of Pediatrics*, 1943, 194-203.

¹⁷ Putnam, T. J., "Mentality of Infants Relieved of Hydrocephalus by Coagulation of Choroid Plexuses," *American Journal of Diseases of Children*, May, 1938, 55:990-999 (references).

lated ventricular system, where it cannot be absorbed, to the subarachnoid spaces where it is absorbed."¹⁸ Through an incision in the temporal side of the skull in front of the ear an opening is made in the ventricle by means of forceps. Of 29 operated cases over one year of age, 24 were "living and cured" after intervals of six months to 23.5 years, of whom



FIG. 32. A marked case of hydrocephaly with a head circumference of almost 21 inches at 8.5 months (left), allegedly cured by an operation before the age of one, which diverted the fluid through an aperture in the third ventricle to the subarachnoid spaces where it could be absorbed. The same girl, mentally normal and well, at the age of 12 in 1945 is seen on the right.*

two were mentally retarded and two were morons. Only one "operative death" occurred. On the other hand, of 63 operated infants under one year of age, only 12 cures were recorded, of whom only five had a normal mentality (see Fig. 32). Of the 12 one was deaf and blind, two had club feet, one was unable to walk, two had convulsions, and seven were "decidedly subnormal mentally."¹⁹ In these infants much

¹⁸ Dandy, "Strictures of the Aqueduct of Sylvius," p. 5.

¹⁹ *Ibid.*, pp. 11-13.

* After Walter E. Dandy.

of the brain injury had, apparently, been done during the intrauterine period. Perhaps it is not possible to open the subarachnoid spaces in such cases. Operations in the infant cases must be made at the earliest moment and are not recommended by Dandy if the head is much distended (say, over 19.5 inches). The most severe antenatal cases are probably born dead or die soon after birth.

The method of air insufflation, used by Frisch for about a quarter of a century without untoward results when properly done, is reported by him to have given excellent results in many cases of "hydrocephalus occultus," but the operation must be repeated at intervals of "several months" until puberty, when the effects may become durable.²⁰

Bucy is frankly pessimistic regarding the value of all forms of surgical interference. "Cases of spontaneous arrest are so common that they make one doubtful of any case reported as cured by any surgical measure." "At the present time even the best intentioned surgery can hardly serve other than to deny to some few cases the possibility of a spontaneous recovery by subjecting them to an operation and a surgical death."²¹ Controversies regarding the value of cerebral and cranial operations must be resolved by the brain surgeons. Admittedly, surgical procedures are futile after extensive, irretrievable damage has been done to the cerebral tissue.

Along prophylactic lines, Votow suggests that the early use of sulfonamide drugs, and possibly penicillin, may prevent the cases that spring from ear and sinus infections.

The education of hydrocephalics

The care of hydrocephalics during the active stage until the acute symptoms have subsided is the responsibility of the physician in cooperation with the hospital or the residential institution and the home until the disease has become non-progressive or stationary through spontaneous recovery or

²⁰ Frisch, "Cases of Hydrocephalus Occultus," pp. 52 f.

²¹ Bucy, "Hydrocephalus," in *Practice of Pediatrics*, pp. 23 f.

cure. Thereafter the education of hydrocephalics of school age becomes the responsibility of the state institution for low grade mental defectives and of the public schools for all those above the low imbecile level who are not in need of institutional care. Questionable lower grade cases should be admitted on probation in special classes. Mental defectives above the questionable lower level should be admitted to special classes for differentiated instruction according to individual requirements. Some should be admitted to opportunity classes or slow sections for remedial instruction and restoration to grade. Those who are mentally normal are admissible to the regular grades although some who are physically frail might fare better in a special class until they regain their physical stamina.

The educational program should be differentiated to meet not only the intellectual level and special intellectual abilities or disabilities and interests, but also the sensory handicaps (visual and auditory) and neuromuscular disorders that may be present. Some require a program of muscle re-education to lessen muscular weakness or clumsiness and to improve motor coordination.

The educational prognosis for hydrocephalics varies greatly according to the native endowment and the amount of cerebral damage wrought before spontaneous recovery or medical cure occurred. Many who are successfully treated before permanent cerebral deterioration occurs make normal educational progress along all lines in which they possess native talent.

ILLUSTRATIVE CASES: The I. Q. of G. M., a case of spontaneous arrest, was 58 by the Stanford-Binet and 52 by the Arthur Performance Scale when she was assigned to a special class in a Wilmington, Delaware, public school at the age of 10.5. She was subject to left internal strabismus. She had not created any serious problem at home except that she was unsteady on her feet and would fall frequently. She had been diagnosed as a hydrocephalic, but had not received any specific treatment for the dis-

order. At the age of 11.3 the head girth was $22\frac{1}{4}$ inches at the supramarginal level and 23 inches over the line of maximum frontal antero-posterior expansion. At the age of thirteen she was rated as doing IIA in reading fluency and comprehension and IIB in spelling. Her best work in the special class was reported in reading and poorest work in handicraft. She was of agreeable disposition, quiet, and orderly. At the age of sixteen the progress report indicated that she had made only "fair" progress and



FIG. 33. O. D., hydrocephalic head, circumference 26.5 inches at age 17.8. Binet age 7.4, I. Q. 46.*

that her greatest capacities were in reading and penmanship. Her poorest ratings were in ability to concentrate, retain, and observe, and in initiative and judgment. She was courteous, honest, and obedient, but inclined to be selfish.

O. D. (Fig. 33), who classifies among the higher grades of mental defectives, had at the time of the examination, at the age of 17.8 years, a Binet age of 8.2 years by the 1908 scale and 7.4 by the 1911, with an I. Q. of 46, and in rate of psychomotor reaction (Seguin form board) measured 6.5 years, according to the author's norms, whether based on the best trial or the average of

* Wallin, J. E. W., *The Education of Mentally Handicapped Children*. Boston: Houghton Mifflin Company, 1939.

three trials. She was very slow in her motor and intellectual reactions. In anthropometric measurements her percentiles were as follows: standing height, eighth; sitting height, thirtieth; weight, tenth; right grip, ninth; and left grip, eighth. Her head was reported to be large at birth. The girth at eighteen months, 18 inches, may have been incorrectly recorded as this is about average for a one-year-old girl. At the age of 17.8 her head, according to the writer's measurement, showed a circumference of 26.5 inches. She did not sit up until eighteen months of age. Prior to this her head had to be propped up with a pillow or be supported by someone's arm. In the special class she was often observed resting her heavy head in her hands with the elbows on the desk. She was almost nineteen years of age when the photograph was taken. So far as known, she had not received any specific treatment for the hydrocephalus. At the age of twenty-two, after a few months in a kindergarten and ten years in a special class in St. Louis, she did about third-grade work in reading and spelling, and second-grade work in oral and written language and arithmetic. She did well in folk dancing, fairly well in running, tactics, and marching, but poorly in mental and physical games, in which she was disinterested and lacked confidence. She did well in writing, brushmaking, plain hand sewing, and simple decorative stitching, and was excellent in sewed and woven basketry which she greatly enjoyed. Her greatest deficiency was in arithmetic, especially problem work, and her greatest capacities in writing and music. Although tiring quickly, she was cooperative and attentive, put forth good effort, but was slow to observe and showed poor judgment. Though cheerful and obedient, she was timid and shrinking, and had always shown lack of self-confidence. No marked motor disabilities were apparent.

Chapter 21

SYPHILITIC BRAIN DEFECTS

Nature of the syphilitic infection

Syphilis (or lues) is a contagious venereal disease produced by a threadlike micro-organism (called *spirochaeta pallida* or *treponema pallidum*, discovered in 1905), which is commonly transmitted from the secretions or discharges of an infected person to another person through the mucus membrane or an abraded surface of the skin. The germ may invade almost any and every tissue of the organism, the skin, mucosa, lymphatics, blood, bones, and nervous system. When the whole organism has become more or less affected, the disease is known as constitutional syphilis; when the central nervous system becomes involved, it is designated neurosyphilis. The manifestations of neurosyphilis are exceedingly multiform, including a well-recognized and serious form of inflammatory degeneration of the brain which (unless arrested by modern treatment techniques) may lead to a specific mental disease (psychosis) known as general paralysis, paresis, or dementia paralytica¹ among adults and as juvenile general paralysis among children. Fortunately, only a small proportion of adult syphilitics, perhaps only 4 or 5 per cent. become paretics, the proportion among men being from two to five times greater than among women, whose nervous systems, apparently, have acquired greater immunity to this form of infection. However, among children the sex ratio is about the same and the incidence amounts to only about 1.5 per

¹ This term was agreed upon in 1930 by the editors of certain journals in psychiatry and neurology.

cent of congenital syphilitics. This discussion of syphilization will be confined largely to the problems created by the prenatal form of infection. Syphilis in young children is predominantly of congenital origin.

Congenital syphilis

Congenital syphilis, although sometimes referred to as heredosyphilis, is not hereditary in the strict sense of the word. It is as truly acquired as is syphilis acquired after birth.² The fetus is directly infected *in utero* by the living spirochete through the placental circulation, or possibly while passing through the birth canal. It is also possible that the poison generated by the pathological organisms may directly affect the sperm and the spermatozoa and also the cortical neurones and may thus interfere with the healthy neural development of the fetus, but this has not been conclusively established. At any rate, the germs have been found in nearly every organ of syphilitic stillbirths. Many of the most severely infected are so debilitated that they are born dead or die in early infancy.

Neural defects. The highly complicated neuropathology of those who survive, as revealed by post mortems, differs very greatly from ultramicroscopic neurone changes to gross brain lesions. Among the latter are multiple diffuse lesions, localized areas of softening, hardening (sclerosis), soft, gummy tumors (known as *gummata*) which are infrequent, inflammation of the innermost lining of the arteries and of the meningeal blood vessels, thickening of the arteries, atrophy of the convolutions, and inflammation of the meninges (meningoencephalitis).³ Any part of the brain and the spinal

² For differences between the prenatal and postnatal forms of syphilis, and for a differentiation between the early, late, and latent stages of the congenital form, see Scheidemann, N. V., *Psychology of Exceptional Children*, II, pp. 405-433.

³ The neurosyphilologists differentiate various kinds of brain syphilis, depending on the structures involved, such as: meningoencephalitic, meningo-vascular, and parietic neurosyphilis. There is doubtless much overlapping between the different forms.

cord and cerebellum may be invaded. The cortex, particularly in the anterior portions of the frontal and temporal lobes, is especially vulnerable to inflammatory degenerative changes. Among the more extensive but unusual developmental anomalies are porencephaly (cavities in the brain), complete lack of brain development (anencephaly), and microcephaly. The concomitants of the gross cerebral defects are occasionally hydrocephaly, and, more frequently, paralysis of one or more limbs (hemiplegia or diplegia; usually one leg) and epileptic convulsions. The latter are particularly prevalent in mental defectives or imbecile and idiot levels.

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Physical signs. Some of the so-called "stigmata" of syphilis are present at birth, some appear within the first two months, and others mainly between the fifth and fifteenth year. In the so-called latent forms the lesions may appear suddenly after a latent period free from obvious clinical signs. The well-recognized "physical stigmata" of syphilis (now less prevalent than formerly) include a saddle nose (depression of the nasal bridge), a skin rash (often of dull red or copper color), Hutchinson teeth (a semilunar indentation or notch with the concavity downward in the cutting edge of the permanent teeth, particularly of the two upper central incisors, and occasionally of the upper lateral and lower incisors), mouth scars (or fissures at the angles of the mouth), enlarged glands, snuffles, hyperactive and asymmetrical reflexes, thickening of the bones at the joints (particularly the elbow joint), prominence of the bones of the forehead; defects of hearing; sore eyes, strabismus, nystagmus, unequal or irregular pupils.

lack of the light reflex (Argyll-Robertson pupil, which responds to accommodation but not to light), partial blindness, and especially interstitial keratitis. The latter is a chronic type of inflammation of the cornea which gives it a ground-glass appearance. Optical atrophy may occur, but less frequently. Deficiencies of vision and hearing are among the

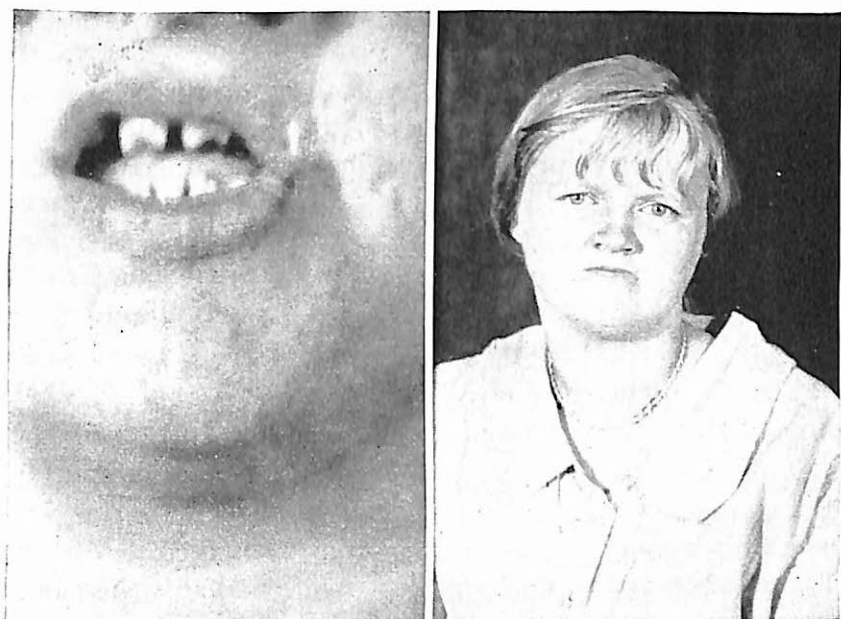


FIG. 34. A 16-year-old congenital syphilitic, showing a 4 plus Wassermann, with Hutchinson teeth, mouth scars, a sunken nasal bridge, and keratitis. Stanford-Binet I. Q. at first test 73, at second test 71, at third test (age 16) 80.

first signs to appear in the delayed form of the disease. Impairment of vision or blindness, however, is sometimes produced by direct gonorrheal infection of the eyes during birth (ophthalmia neonatorum). This form of eye defect is readily preventable by the prompt depositing of a drop of one per cent solution of silver nitrate in each eye of the newborn.

Fig. 34 shows some of the classical physical stigmata of congenital syphilis.

In rare instances, juvenile tabes may result at about the

age of fifteen from an invasion of the spinal nerves, producing, among other things, an unsteady gait, incontinence, inequality of the pupils, and failure of the pupils to react to light.

Some congenital syphilitics manifest the gross signs of syphilis, others do not; some give a positive Wassermann, others do not. No single gross "stigma of syphilis" can be regarded as conclusively pathognomonic of syphilitic infection. The stigmata are merely suspicious signs. A multiplicity of signs is more indicative of the presence of syphilis than one or two. Confirmation of the diagnosis should be sought from three sources: (1) similar suspicious signs in the siblings; (2) a record of sterility, miscarriages, or stillbirths in the mother; and (3) positive serological tests or other tests administered to the patient, the parents (particularly the mother), and the siblings. The best known among these tests are the Wassermann test of the blood and spinal fluid, and the Noguchi and the Kolmer modifications of the Wassermann; the Kline-Young, the R. L. Kahn, the Meinicke, and the Sigma precipitation or flocculation tests; and the R. L. Lange test (gold-sol or colloidal gold test) for the presence of protein-globulin in the cerebrospinal fluid for the diagnosis of cerebrospinal syphilis. Unfortunately these delicate tests sometimes give discrepant results.

The routinely used test, the Wassermann, although more definitely diagnostic when it is three or four plus, does not yield unambiguous results. For example: more positive results are obtained in childhood than during adulthood, and many who show positive reactions in early life become negative as time goes on (Harry C. Solomon and Charles S. Woodall), even without treatment, apparently as a result of spontaneous immunization through the development of antibodies or the inactivation of the germs in some other manner. The reactions of congenital adult syphilitics are usually negative. On the other hand, infants who show negative Wassermans at birth may later give positive reactions (Philip C. Jeans and

Jean V. Cooke). Low grade cases (idiots and imbeciles) often show negative reactions in the presence of clear clinical stigmata (Benda).⁴ Infections other than syphilitic may also produce positive Wassermanns. The disease is, moreover, very capricious in its occurrence, affecting some siblings but not others, even one of the monovular twins but not the other. Apparently some persons may be congenitally more resistant than others to the infection or may have acquired immunity.

The classical stigmata of congenital syphilis have, according to R. M. Stewart, become uncommon during the present century, possibly because the infection has grown milder or greater resistance or immunity has been acquired. "The more usual clinical signs, such as rhagades (chaps in the skin), Hutchinsonian teeth, and eye changes, form a clinical picture which is only seldom met with, and statistics based on the presence or absence of the clinical signs will lead to a very erroneous idea of the incidence of the syphilitic taint."⁵ Only 2 of 173 positive Wassermanns exhibited the classical triad of notched incisors, eighth-nerve deafness, and keratitis.

Course of the malady. The child may seem to be normal at birth, or he may be underweight and appear wizened, anemic, and debilitated. At the age of one or two growing paralysis of one or more limbs or of an eye muscle (strabismus) may become apparent. As the paralysis gradually gets worse, epileptic seizures may develop and the child may begin to deteriorate mentally rather conspicuously, especially with respect to retentiveness and judgment. The less severe cases often show developmental retardations, such as delays in speech and a stationary or falling I. Q. The over-all picture of the uncured cases is one of progressive deterioration and infantilism, bodily and mental.

Mental signs. Just as the child may suffer from congenital

⁴ Benda, C. E., "Congenital Syphilis in Mental Deficiency," *American Journal of Mental Deficiency*, July, 1942, 40-48.

⁵ Quoted from Penrose, *Mental Defect*, p. 120.

syphilis without showing any gross clinical signs, so may be free of any obvious mental stigmata and pass muster as mentally normal, especially if the microbes have not invaded the brain or have become inactivated by early treatment. The distribution of intelligence will vary from supernormality to idiocy. Based on the performances in the Binet-Simon scale of 45 syphilitic children in a children's clinic and a hospital in Budapest, ages three to sixteen, Paul Von Kiss and Tibor Raja concluded that over twice as many syphilitics tested "retarded" as did 342 nonsyphilitic school children in the same city. Only 6 per cent of the latter tested at least two years backward as compared with 28.9 per cent of the syphilitics. Only 11 per cent of those who received anti-syphilitic treatment during infancy tested retarded as compared with 58.8 per cent of those who were treated between the second and thirteenth year.⁶

The number of children in institutions for the feeble-minded who are syphilitic and the number of children who are mentally defective or subnormal to a lesser degree because of syphilitic infection are still debatable because of the contradictory nature of the findings. The average incidence of syphilis in institutions for mental defectives based on the examination of 24,313 inmates by many examiners is 4.83 per cent,⁷ which is only about half as much as the incidence of positive Wassermanns among 20,647 pregnant women examined in hospitals, clinics, and private practice in different parts of the country. However, as already emphasized, many of the severely infected are born dead or die in early infancy, and many of the low grades react negatively in spite of obtrusive signs of lues. Moreover, the Wassermann does not reveal the latent forms. Although some syphilitics are probably primary types of mental deficiency, it is doubtless also true that others owe their mental defects to the cerebral de-

⁶ Cited from Scheidemann, *The Psychology of Exceptional Children*, pp. 423 f.

⁷ Hays, Robert R., "The Role of Syphilis in the Production of Mental Deficiency," *Journal of Psycho-Asthenics*, 1939, No. 2, 25-33.

generation, and possibly germinal deterioration as well, caused by this virulent infection.

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Shuttleworth and Potts, *Mentally Deficient Children*, pp. 146-147.

Tredgold, *Mental Deficiency*, p. 270.

Wallin, *Problems of Subnormality*, pp. 441-444.

The majority of the survivors whose brains have been seriously "eroded" by the disease and who are subject to seizures and paralyses (and sometimes hydrocephalus as well) are of unmistakable idiot or imbecile grade.⁸ The earlier the appearance of the morbidity and the greater its severity, the greater is the intellectual and temperamental disorganization.

In the writer's experience many juvenile syphilitics who test merely intellectually backward do not make as much progress as the primary type of moron of a lower Binet level. Many of them are more or less infantile, puerile, emotionally or temperamentally unstable, flighty, highly distractible, incapable of sustained concentration, and incapable of making the educational progress one would expect from their rating in intelligence tests. The educational work for syphilitics needs to be adjusted to their educational, intellectual, physical, temperamental, and sensory (visual and auditory) disabilities. Although many adjust properly in the regular grades and are capable of making normal educational progress, others need the individual assistance afforded by a special class, even some who would not be assigned on the basis of a low Binet I. Q.

Some syphilitic children, as already intimated, constitute

⁸ Benda, "Congenital Syphilis in Mental Deficiency," p. 45.

distinct behavior problems.⁹ Subject to temperamental outbreaks and moral obtuseness, as some of them are, they may indulge in the general run of petty delinquencies and also grosser offenses, such as sexual misconduct. The program of socialization, character education, habit formation, and preventive and re-educational work that many require should be based on adequate physical, psychiatric, and psychological studies.

Juvenile general paralysis

A few children antenatally or postnatally infected with syphilis who may have shown no evidence of gross brain lesions may, nevertheless, undergo a process of serious progressive mental and physical degeneration. The mental changes usually develop slowly and insidiously until the physical signs appear at about the onset of puberty. Thereafter the disorder (named juvenile general paralysis by Thomas S. Clouston in 1877) may become rapidly progressive and may reach a fatal termination in some cases within a year or so and in other cases within five or six years. The child previously mentally normal or subnormal, as the case may be, becomes dull and apathetic, loses interest in his studies, fails to make as much progress as he used to do, and begins to deteriorate. One of the first signs of scholastic deterioration is the loss of the most recent acquisitions, such as writing, arithmetic, and reading. The child may also undergo changes in disposition and become seclusive and depressed, or irritable, boisterous, or excitable, and subject to emotional tantrums and petty delinquencies. Gradually motor disabilities become prominent, such as stumbling or motor incoordination in walking, speech disturbances (hesitant, slurring, and indistinct speech), writing disturbances (angularity, tremors, misspellings, duplications, or omissions), irregular, dilated pupils, loss

⁹ Jenkins, Richard L., and Crudim, Myrtle, "Behavior Problems in Children with Syphilis," *American Journal of Orthopsychiatry*, October, 1941, 752-769.

of the light reflex, fibrillary trembling of the fingers, tongue, and lips, growing spastic paralysis, exaggerated reflexes, and possibly convulsions. Thereafter the victim may become increasingly stupid, indifferent, dirty in his habits, emaciated, and eventually bedridden with contracted limbs. When the infection is very virulent or the cerebral neurones nonresistant, the varied classical symptoms of the third stage of adult paresis tend to develop and the child becomes a helpless dement before death supervenes, often in a coma. Remissions and hallucinations and delusions are less common in the juvenile than in the adult form of paresis.

Medical treatment of syphilitic infection

The microbe of syphilis has through the ages been one of the most virulent of the pathogenic bacteria, one of the "greatest slayers of the unborn," which has largely remained immune to disinfections until recent times. Under modern antisyphilitic treatment ordinary cases of syphilis can now be successfully treated provided irreversible damage has not already been done, by the administration of mercury, organic arsenicals, or salts of bismuth. The well-known 606 or salvarsan of Paul Ehrlich fame contains arsenic. Favorable reports have also been forthcoming from the use of penicillin.¹⁰ Complete cures often require many months of treatment.

These forms of treatment have not proved very effective with the gravest form of neurosyphilis, namely, paresis. Although the reports are somewhat discrepant, adult paresis, if treated persistently in the early stages (and occasionally in the later stages), can in many cases be mitigated to a considerable extent, and some cases can be restored to economic efficiency and be released from the institution. The modern treatment involves the use of certain drugs, such as an arsenical (tryparamide) and bismuth salicylate, and some form of fever ther-

¹⁰ Dattner, Bernhard, *Penicillin in Neurosyphilis*. New York: Grune & Stratton, 1948. Herrell, Wallace E., *Penicillin and Other Antibiotic Agents*. Philadelphia: W. B. Saunders Company, 1945.

apy. High body temperatures (from about 104° to 107°) prove germicidal for the syphilitic micro-organism. The earliest method for elevating the body temperature was by means of malariotherapy or inoculation with malaria germs (first used by Julius Wagner-Jaurezz, the Austrian psychiatrist, about 1917), followed by injection with typhoid vaccine, apparently less successfully. More recently the fever has been produced by the use of air conditioned fever cabinets with metal electrodes attached to the patient (the Burdick diathermy method), or by the Kettering hypertherm cabinet, heated by an electric contrivance. Or the patient may be covered by blankets, placed between condenser plates, or be subjected to short radio waves.

All of these fever treatments are strenuous and cannot be used with patients suffering from serious heart, kidney, or lung diseases. They are, according to report, less effective with juvenile than with adult paresis.

A program of education is scarcely admissible for juvenile paretics who are undergoing active deterioration; they require custodial care or hospitalization.

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Paresis to Hospitals for Mental Disease in New York State, Year Ended March 31, 1945," *Psychiatric Quarterly*, 1947, 21:212-232 (the admission rate is declining).

The communicability of syphilis and the problem of prevention of venereal infection

Syphilis is highly infectious during the primary and secondary stages. The latter stage, characterized by fever, multi-form skin eruptions, iritis, and severe pains in the head, joints, and membranes covering the bones, develops between the sixth and twelfth months. But even during this period the infectiousness can be curbed after one month of regular weekly injections. So treated, a child can return to school or a worker to his job without danger of transmitting the infection. The later stages, or the latent or noninfectious forms, of syphilis are not dangerous from the point of view of contagiousness. The disease becomes noninfectious after a period of years (five or more) even without treatment. Some authorities believe that if the mother is subjected to antisyphilitic treatment before the fourth month of pregnancy the child will be born free of infection.

Here, as in many other fields, the paramount problem is prevention of venereal infection rather than cure. The experience of the Scandinavian countries indicates that the incidence of syphilis can be greatly reduced by the requirement of blood tests before marriage and enforced treatment when needed. The use of venereal prophylactics will prevent syphilitic and gonorrheal infection to a very high extent, although some object to the dissemination of information concerning prophylactic measures because of the fear that the information will encourage venery and lead to moral degradation.

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